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THE DISTINCTION BETWEEN CHRONIC GLOMERULONEPHRITIS AND HYPERPIESIA (PRIMARY HYPERTENSON)

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Chronic glomerulonephritis and hyperpiesia are frequently confused clinically, though they are entirely distinct diseases. The failure to make the distinction is usually due to the fact that both conditions commonly have the same main features, viz., high blood pressure and abnormal urine. The object of this paper is to call attention to the fundamental differences in pathologic anatomy and to indicate how the distinction can usually be made on clinical data.

Moderate chronic hypertension is characteristic of hyperthyroidism and is occasionally found in bilateral hydronephrosis and congenital cystic kidneys. A marked hypertension occurs in the toxemia of pregnancy. Hypertension may occur acutely from urinary obstruction, from increased intracranial pressure (brain tumor, hemorrhage, or from excitement, etc.). A high systolic (with low diastolic) pressure is frequent in aortic regurgitation. But, aside from these conditions, which are usually easily recognized, hypertension is to be referred to hyperpiesia or to glomerulonephritis.

Glomerulonephritis is an infection of the kidney, usually streptococcic, in which the glomeruli are primarily and chiefly affected. The bacteria in the circulating blood are phagocyted and destroyed by the endothelium of the glomerular capillaries, but the irritation causes these cells to enlarge and proliferate, resulting in closure of the capillaries. This proliferative reaction is the fundamental and most important process in glomerulonephritis. There are other features of less importance: the formation of epithelial crescents from Bowman's capsule, which compress the tufts; the presence of polymorphonuclear leucocytes in the glomerular capillaries and to some extent outside the corpuscles; and the injury of the tubules. There is seldom necrosis of any cells and practically all the changes that occur in the kidneys subsequent to the infection are to be referred to the effects of closure of the glomerular capillaries. It is true that there is always some tubular injury and often a little interstitial exudate, but these changes are unimportant in the subsequent course of the disease.

When nearly all the capillary bed of a glomerulus is closed, the blood can no longer pass through, and the associated tubule undergoes disuse atrophy to the point where it joins the collecting tubule. When a number of glomeruli in different parts of the kidney are closed the result is an uneven cortical atrophy resulting in a granuular surface. Where the tubules disappear there is a replacement fibrosis often with a few lymphocytes, an appearance responsible for the old term "interstitial nephritis."

The great majority of cases of acute glomerulonephritis terminate ultimately in complete recovery, a few die and a few pass on into the chronic form. In the fatal cases we know that there is occlusion of practically all the glomeruli. In the cases that recover we have evidence, derived from the study of mild cases dying of some other disease, that only a part of the capillary bed is permanently occluded. Acute glomerulonephritis is readily traceable in most instances to some infection, scarlet fever, tonsillitis, etc. The characteristic features are: (1) abnormal urine (albumin, casts, erythrocytes); (2) usually edema, which is often severe; (3) often a moderate hypertension, especially early in the attack; (4) renal insufficiency in the terminal stages of fatal cases. Acute glomerulonephritis is synonymous with acute Bright's disease and is readily recognized clinically.

Subacute glomerulonephritis (synonymous with subacute Bright's disease) is not sharply separable from either the acute or the chronic form. A case can hardly be called subacute until the ultimate outcome is apparent. By arbitrary definition it is commonly employed for cases that begin acutely and are continuous to a fatal result within one year.

The outstanding features are: (1) abnormal urine; (2) edema, usually; (3) hypertension, moderate, but more pronounced than in the acute form; (4) renal insufficiency, which usually becomes more pronounced. At postmortem the kidneys are usually enlarged, cloudy and swollen, with smooth external surfaces. The capillaries of the glomeruli are practically all more or less completely closed and there is widespread tubular atrophy. There has been insufficient time for cortical shrinkage. Apparently all the glomeruli were severely injured in the initial infection. This form of nephritis is not confused with hyperpiesia.

Chronic glomerulonephritis is not synonymous with chronic Bright's disease, but includes one form of this rather ill-defined group. Only an occasional case is traceable to a definite acute attack. The onset is usually slow and gradual and the patient commonly comes to his physician in a well advanced stage of the disease. The early stages have not been well studied, since they are only detected occasionally and by accident in examinations for life insurance, or other purposes. The prominent symptoms of a well developed case are weakness, dyspnea on exertion, pallor, headache, frequent urination, edema, epistaxis, etc.

The urine is practically never entirely free from albumin, although the amount varies widely. Casts vary with the albumin. Erythrocytes are usually present and are especially abundant during exacerbations. In cases of long duration the specific gravity tends to become fixed at a low level and the quantity of urine increases. During exacerbations and in terminal stages there is oliguria.

The blood shows a progressive secondary anemia. Severe anemia is evidence of an advanced stage of the disease.

The blood pressure is almost invariably increased, the systolic usually ranging from 150 to 200. In cases of many years standing it may be higher, but pressures above 240 are apparently very rare.

The heart shows a moderate hypertrophy of the left ventricular type, but since it is not dilated the measurements are not greatly increased. Cardiac decompensation is very unusual.

Functional tests are useful in the diagnosis of advanced cases, but are usually disappointing in the earlier stages. The phenolsulphonephthalein test is perhaps the most delicate, if freed from technical errors. Retention of urea nitrogen and creatinin is a late manifestation. The Volhard concentration test is simple and gives about the same information as the others.

Death occurs in most instances from renal insufficiency, occasionally from edema of the lungs, rarely from an intercurrent or terminal infection such as pneumonia.

The kidneys are often small and contracted (secondary contracted kidney, chronic interstitial nephritis), but they may be normal in size or even enlarged (chronic parenchymatous nephritis). It is seldom possible to predict the size of the kidneys from the clinical data, but in a general way large kidneys are more often associated with heavy albuminuria and marked edema.

Microscopically, one finds large numbers of hyaline glomeruli whose tubules have entirely disappeared. Other glomeruli are enlarged from endothelial swelling but some of their capillaries are still permeable and their tubules are intact or only partially atrophic. Very few are normal. Renal function is carried on toward the last by damaged glomeruli with a variable number of open capillaries. In the large kidneys there are fewer hyaline glomeruli and atrophied tubules. The damaged glomeruli are often associated with tubules in which the cells are swollen and contain abundant lipoid droplets. The cause of this peculiar change is not understood. The usual kidney that one sees is intermediate between the typical contracted (interstitial) and parenchymatous types. Whatever the gross appearance of the kidney, it shows the fundamental feature of glomerulonephritis, viz., inflammatory occlusion of the glomerular capillaries. The arterioles of the kidney are seldom involved except in cases of very long duration in which they may show some hyaline degeneration.

The clinical course of chronic glomerulonephritis is often marked by exacerbations following colds, sore throat, etc. Each infection seems to decrease the permeable glomerular capillaries and bring the patient nearer to renal insufficiency.

Hyperpiesia (primary, or essential hypertension) is chiefly a disease of advanced life. In a group of 200 cases from our postmortem records 89 per cent were over forty years old, and 73.6 per cent were over fifty years. It is apparently over twice as common in males as in females. Hypertension may exist without subjective symptoms. Presum-

ably there is this latent period in most chronic cases, but we have no accurate knowledge of its duration. The first symptoms may be nervousness, irritability, insomnia, headache, nocturia, etc. As the disease progresses symptoms referable to the heart, brain or kidneys may develop.

Two hundred and eighteen cases that came to necropsy may be subdivided on the basis of clinical course and cause of death as follows:

I. Hyperpiesia with cardiac symptoms....			155
A. Myocardial exhaustion		98	
B. Coronary sclerosis		35	
a. preceded by cardiac symptoms...	15		
b. sudden death	20		
C. Brain lesions		6	
a. hemorrhage	1		
b. thrombosis	4		
c. undetermined	1		
D. Renal insufficiency (a prominent factor)		7	
E. Accident		2	
F. Intercurrent diseases		7	
II. Hyperpiesia without prominent cardiac symptoms			63
A. Brain lesions		32	
a. hemorrhage	23		
b. thrombosis	5		
c. undetermined	4		
B. Renal insufficiency		9	
C. Accident		10	
D. Intercurrent diseases		10	
E. Undetermined		2	

I. *Hypertension with cardiac symptoms.* — A. Myocardial exhaustion. Ninety-eight patients suffered chiefly from cardiac decompensation and died apparently from myocardial exhaustion. This type of hyperpiesia is often called "chronic myocarditis." The first symptoms to attract the patient's attention are usually dyspnea, especially on exertion, and edema. The heart shows hypertrophy of the left ventricular type, and, when the left ventricle begins to fail, enlargement of the right heart, also. Relative mitral insufficiency is common; relative aortic insufficiency rare. The blood pressure is usually high, the systolic often being over 200. Sometimes, however, the pressure is normal when the patient enters the hospital. The case with a low blood pressure may be difficult to distinguish from an old valvular defect unless a previous record of hypertension is available. The shape of the heart as seen in the roentgenogram is helpful in the differential diagnosis.

The liver is often palpable and tender, and the spleen may be palpable. There may be physical signs of congestion and edema of the lungs. Remissions of varying duration occur frequently.

The urine commonly shows albumin and casts; but frequently it is normal, especially when the heart is compensated. Functional tests usually show normal values except the phthalein test, which is frequently low (30 to 40 per cent) chiefly because of passive congestion of the kidneys.

The blood is normal, or the erythrocytes and hemoglobin may be increased.

B. Coronary sclerosis. Twenty patients died suddenly of coronary sclerosis, without having had any prominent symptoms previously. In fifteen others there was a history of decompensation with anginal attacks, and death was due mainly to coronary disease.

C. Six patients with cardiac decompensation died of apoplexy.

D. In seven patients with cardiac decompensation renal insufficiency was the chief cause of death. This was determined either clinically by functional tests, or by the extensive renal atrophy found at postmortem.

II. *Hypertension without prominent cardiac symptoms.*—In this group there is little or no clinical evidence of myocardial exhaustion and no passive congestion of the liver postmortem. The most common cause of death is apoplexy. Nine patients died of renal insufficiency. Many die of accidents and of intercurrent diseases, such as pneumonia and carcinoma. The urine is frequently normal except in those with renal involvement. The erythrocytes and hemoglobin are normal or increased except in a few cases with marked renal insufficiency in which there may be secondary anemia.

Postmortem findings. *Heart.* Left ventricular hypertrophy is constant. In cases with prolonged decompensation the right ventricle is also enlarged and all the chambers are dilated. The coronary arteries show atherosclerosis, usually somewhat more advanced than the average for this age group, and in thirty-five cases coronary disease was so pronounced that it was considered the immediate cause of death. The myocardium shows hypertrophy but no other changes except those attributable to coronary disease. The mitral valve frequently shows a demonstrable relative insufficiency from dilation of the heart chambers. *Kidneys.*

Aside from passive congestion the kidneys are usually fairly normal on gross examination. However, slightly granular external surfaces with adherent capsules are frequently seen. Kidneys with an appreciable gross atrophy were found in sixteen cases and of these only eight were typical primary contracted kidneys. This form of contracted kidney is also called chronic Bright's disease. There are all transitions from typical contracted kidneys to those that appear entirely normal. Microscopically in the kidneys with gross contraction there are areas of atrophy corresponding to sclerotic arteries and arterioles. There are many hyaline glomeruli with atrophic tubules. The atrophy is due to closure of small arteries and arterioles. The glomeruli show no changes except atrophy. There is no endothelial proliferation or crescent formation, such as are found in glomerulonephritis. *Arteries.* In the small arteries one finds a marked thickening of the intima due to an increase of elastic tissue. The media undergoes atrophy. These small arteries assume the structure of large arteries such as the carotids. The arterioles show typically a deposit of hyaline under the intima with a resulting stiffening of the wall and narrowing of the lumen. The muscular layer undergoes atrophy. These changes in the arterioles are frequently found in kidneys that show no gross or microscopic atrophy. A diagnosis of hyperpiesia may be made from microscopic examination of the kidneys when this lesion is present.

But not all kidneys show arterial disease. In our 218 cases the arterial changes may be classified as follows:

Involvement of small arteries	Per cent
Severe	64.8
Moderate	16.6
Slight	18.1
No disease	0.5

Involvement of arterioles	Per cent
Severe	57.7
Moderate	18.5
Slight	12.7
No disease	16.1

The degree of involvement of the small arteries and arterioles has no relation to the size of the heart or the known duration of the disease. No case of marked renal arteriolar sclerosis was found in an examination of the kidneys from 4,000 necropsies except in cases of hyperpiesia; but, as noted above, many cases of hyperpiesia show no renal arteriolar sclerosis.

The arterioles of the brain and pancreas often show sclerosis, but not so frequently as those of the kidney. Clinical examinations indicate that the retinal arterioles are involved as often as the renal.

The principal differences between chronic glomerulonephritis and hyperpiesia may be given in tabular form.

Chronic glomerulonephritis—
- Age—44 per cent over 40 years
- Blood pressure—Usually moderate hypertension
- Size of heart—Moderate enlargement
- Cardiac decompensation—Very rare
- Secondary anemia—Constant in advanced cases
- Renal function—Low in all advanced cases
- Urine—Always abnormal
- Eyegrounds—Arteriolar sclerosis rare
- Kidneys—Glomerulitis

Hyperpiesia—
- Age—89 per cent over 40 years
- Blood pressure—Usually marked hypertension
- Size of heart—Marked enlargement
- Cardiac decompensation—Present in over 60 per cent
- Secondary anemia—Only in some of renal type
- Renal function—Good except in renal type (8 per cent)
- Urine—Often normal
- Eyegrounds—Usually arteriolar sclerosis
- Kidneys—No glomerulitis; usually arteriolar sclerosis

INTERSTATE ASSEMBLY

This department is all excited over the presence in town of hundreds of visiting doctors.

When one pauses to think how much any and all of them know about one's insides,—how easily they could blueprint one's entire internal economy,—one wonders whether these learned gentlemen do not, after all, regard the individual human being more or less as a collection of giblets enclosed in glass.

A pretty thought, at least.

As for their several and collective vocabularies, the average equipment in this respect simply swoons before it.

Not only is the extent of their terminology positively staggering; but its confusions (to the lay mind) are endless.

Thus we live to learn that phlebitis is not caused by the bites of fleas, but by—well, by something else. Psoriasis, it appears, has no connection whatever with sore eyes, and the diplococcus, mean as he is, is not responsible for the ailment known as diplopia.

It all goes to show how foolish it is for the crass amateur to imagine that he knows anything about anything that ails him. Yes, bichloride, it is! — Frances Boardman, St. Paul Dispatch.

THE ETIOLOGY OF THE CHRONIC HEART MUSCLE DISEASES OF FREQUENT OCCURRENCE IN GENERAL PRACTICE*

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When I began teaching medicine at the University of Minnesota some four years ago I found that the senior students had been impressed with the notion that chronic heart muscle disease not associated with primary valve defect (chronic endocarditis), adhesive pericarditis, or syphilis was largely attributable to chronic foci of infection in the teeth, tonsils, sinuses, etc. Most of these students thought of the heart muscle disease as myocarditis, a name which implies inflammatory processes usually of infectious origin. At the same time I found that many of the general practitioners with whom I came in contact believed very firmly that foci of infection were largely responsible for heart muscle disease in failing hearts not exhibiting signs and symptoms of primary valve defect, adhesive pericarditis, or syphilis. It has been possible to impress upon our students that there is no convincing evidence for this view and much good evidence for other factors in the production of chronic heart muscle disease, so that today very few of them attribute much importance to foci of infection as a principal cause of chronic heart muscle disease, at least in our examinations and quizzes. On the other hand, I find that the general practitioner still believes very firmly that foci of infection are largely responsible for chronic heart muscle failure in hearts in which there has been no previous valvulitis (endocarditis), adhesive pericarditis, or syphilis.

It is of considerable importance to know exactly what are the factors involved in the causation of a disease, for it may be that we can learn how to eliminate or to modify these factors ultimately in such a way as to increase the expectancy or comfort of persons tending toward the disease. I have been engaged upon studies in the etiology of chronic heart muscle disease for some years and I shall attempt to give you a short résumé of the evidence for and against various etiologic factors.

We find at autopsy that the larger proportion of hearts of persons who have died of chronic heart muscle disease show only an increase in weight largely due to hypertrophy of the left ventricle. In a few cases hypertrophy of the right heart only is present. Sometimes they also show moderate or marked degrees of necrosis of fibres with scar tissue replacement. This fibrosis is found in 20 per cent of the hearts showing left ventricular hypertrophy, as well as in an equal number of hearts in which there is no hypertrophy. A few hearts show only fatty degeneration of the fibres, and a very few may show a chronic myocarditis consequent upon an acute myocarditis.

If we take the hearts showing only cardiac hypertrophy, mostly of the left ventricle, we find that the hypertrophy can be explained only upon the basis of increased blood pressure. Hypertrophy is due to increased work and there are only three variables in the equation for heart work: minute volume, blood pressure, and velocity. The latter may be neglected, excepting in severe anemias. Minute volume increase would lead to equal hypertrophy of both chambers. Therefore, we have only high blood pressure in the systemic circulation to explain the left ventricular hypertrophy. Moreover, examination of the kidneys of these cases will nearly always show some evidence of arteriolar sclerosis and this we know to be a lesion associated with hyperpiesia, a disease in which high blood pressures are present for many years before heart failure results. For these hearts, then, and they represent the majority of hearts found in those who have died of chronic heart muscle disease, high blood pressure of many years' standing is the one outspoken factor in causing myocardial exhaustion and failure insofar as pathology throws any light on the subject.

In this country apparently only a very small number of hearts will show right ventricular hypertrophy exclusively. These are cases in which there is at the same time evidence of severe pulmonary disease as emphysema, chronic bronchitis, or long-standing, severe bronchial asthma. Here again the increased work factor is hypertension; hypertension in the pulmonary circuit due to increased resistance in the pulmonary circulation. In Europe these hearts are more common than in this country.

*Read before the Southern Minnesota Medical Association, Owatonna, Minnesota, May 18, 1925. From the Department of Medicine, University of Minnesota.

The second group of hearts, those with scar tissue formation, are practically always found in cases in which the coronary vessels are diseased and wholly or partially obstructed.[1,2] In fact, the scars are the result of the usual scar tissue replacement of fibres killed by anemic necrosis consequent upon partial or total coronary artery or arteriole closure. The evidence from pathology in these cases is for coronary disease as a prominent factor in chronic heart muscle disease. About one-half of these hearts also show left ventricular hypertrophy and intima disease of the small renal vessels. In these cases we may therefore say that high blood pressure and coronary sclerosis are two factors of great importance in the production of heart failure in these hearts.

The third group of hearts, those in which only fatty degeneration is present and excluding all cases of acute heart disease with fatty degeneration, is small in number, perhaps not more than 5 per cent of all the hearts of chronic heart muscle disease cases. It also accompanies pathological evidence of hypertension and coronary disease in some cases. The causes of the fatty degeneration can not always be found. Sometimes it is chronic alcohol poisoning; sometimes, phosphorus or arsenic poisoning; sometimes it is due to anemia; sometimes it is associated with severe infections which have been terminal events. It is possible that chronic foci of infection do at times lead to fatty degeneration of the heart muscle, but up to the present this has not been proved.

We can sum up the pathological evidence as to the factors concerned in chronic heart muscle disease as follows: hypertension is the important factor in the largest number of cases; coronary disease is the important factor in a large number—approximately one-fourth as many as hypertension not associated with coronary disease; hypertension and coronary disease are associated together in about as many cases as coronary disease alone; and various toxic agents, as yet frequently unknown, may cause a small percentage of chronic heart muscle disease. Chronic myocarditis is a rare disease.

Now let us examine the clinical evidence. Seventy to 75 per cent of all cases of chronic heart muscle disease seen by me at the University and at the Minneapolis General Hospital have blood pressures above 170 systolic and 100 diastolic. The majority have blood presures above 200 systolic, and where careful records have been kept these pressures were present for at least four to ten years. If we remember that these patients are bed patients in mild or severe decompensation and also remember that blood pressure tends to fall in hyperpiesia and may even reduce to normal when the patient is put to bed, I think it is no exaggeration to say that high blood pressure is an important factor in producing chronic heart muscle failure in 75 per cent of all cases of so-called "myocarditis." A calculation shows that hearts sustaining blood pressures over 240 mm. Hg. do more work per day than the hearts of athletes training for and taking part in severe competitive games; and the hypertension heart may do increased work every day in the year for ten to fifteen years. After some years of this the left ventricle slowly dilates and symptoms of dyspnea develop, often very slowly and insidiously.

X-ray examination gives very good confirmatory evidence of the statement that high blood pressure causes heart failure. When a chamber of the heart becomes insufficient the intraventricular pressure in that chamber rises and the chamber tends to dilate. The pericardium tends to prevent dilatation at first but ultimately the pericardium stretches and demonstrable dilatation of the heart chamber accompanies it. The *x*-ray outline of left ventricular dilatation is typical in the "mean normal" heart. We get a figure suggesting a sock. Only aortic valve disease and hypertension give this *x*-ray figure. It is usually very easy to exclude aortic disease by means of physical signs. A sock-shaped *x*-ray outline of heart, where aortic disease is excluded, means hypertension heart. A large number of hearts in patients with chronic heart muscle disease give this *x*-ray outline. Not infrequently we find this shape of heart in heart failure and at the same time the blood pressure is not elevated. Studies of our cases have shown in many of these cases a previous record of hypertension. We have records of cases exhibiting blood pressures over 200 systolic for three and four years, yet at subsequent examinations showing normal blood pressures. Our studies of such cases justify the statement that high blood pressures or hyperpiesia in from 5 to 10 per cent of all cases fall to normal or

[1] Aschoff: Die hentige Lehre von den pathologisch-enanatomischen Grundlagen, der Herzschwäche. Jena. 1906.

[2] Clawson: The Myocardium in Non-Infectious Myocardial Failure. Am. Jour. Med. Sci., CLXVIII, 648, 1924.

near normal after being elevated for years. For this reason we believe that hypertension is an etiologic factor in even more than 75 per cent of all chronic heart muscle disease.

Further evidence that some cases of heart disease due to hypertension show no elevated blood pressure at the time of hospital examination is found at autopsy. We have seen cases of heart failure with normal blood pressure die and come to autopsy where the pathological evidence of previously existing hypertension was found.

The very best evidence that not all cases of hypertension heart show elevated blood pressures at the time of examination is found in the eye ground examination. We are now in position to diagnose hyperpiesia (essential vascular hypertension) with surety from the ophthalmoscopic findings. The large majority (80 per cent) of hyperpiesia cases in late stages show pathologic processes in the retinal vessels comparable to those seen in the kidneys. We frequently see cases in which the blood pressure is normal at the time of examination, yet the eye ground study gives unequivocal evidence of previously existing high blood pressure. Such studies have convinced us that even more than 70 to 75 per cent of all chronic heart muscle disease is or has been associated with high blood pressure.

By means of the *x*-ray we have followed some of our cases of hyperpiesia from a stage in which the heart shows no *x*-ray evidence of left ventricular dilatation into stages of increasing dilatation and finally decompensation. These hearts may dilate considerably before clinical evidence of decompensation is evident. A permanent drop of blood pressure is not often seen, but I have seen a few cases. In these cases the progressive dilatation stopped and even reduced in extent and the mild clinical symptoms of decompensation disappeared —in one of my cases disappeared for eight years.

Summing up: clinical evidence indicates that at least 75 per cent of all chronic heart muscle disease is associated with high blood pressure and the high blood pressure is at least one very prominent factor in the left ventricular dilatation and heart failure.

Clinical evidence for coronary sclerosis is not as frequently obtained in cases of coronary sclerosis as clinical evidence of hyperpiesia is obtained in cases of hypertension heart. *Attacks* of angina pectoris, *attacks* of cardiac asthma, especially *attacks* of nocturnal cardiac asthma, and a negativity of the T wave in leads I and II are not infrequently evidence of coronary disease, but many cases of coronary sclerosis come to post-mortem with no clinical evidence of disease. Post-mortem evidence is the best indicator we have of the incidence of this type of chronic heart muscle disease. The autopsy material of the Pathological Department of the University would lead us to believe that approximately 15 per cent of all cases of chronic heart muscle disease are associated with coronary sclerosis alone; about an equal number are associated with both hypertension and coronary sclerosis.

We can therefore account for the etiology of about 90 to 95 per cent of all cases of chronic heart muscle disease. At the present moment there are about 5 to 10 per cent of all cases for which we have difficulty in assigning well established etiology. Some of these cases can be accounted for as syphilis of the myocardium, an etiology stressed by Warthin and proved by him for some cases. Chronic intoxications of various kinds will account for some of these cases. No one can deny that chronic foci of infection may account for some of these cases, but we need proof of it. Certainly true chronic myocarditis is rarely seen by the pathologists and fatty degeneration of the heart not associated with hypertension or coronary disease is also not very common.

Moreover, no one can deny that chronic foci of infection may be an adjuvant etiologic factor in the production of heart failure in cases of hypertension heart and in cases of coronary sclerosis. At present there is no good evidence for this. To shoot a few cubic centimeters of a broth culture containing streptococci into a rabbit is to produce a severe septicemia. Lesions thus produced in the heart muscle are either of the nature of abscesses or acute toxic myocardium or acute interstitial myocarditis, and are not analogous to the lesions seen frequently in the chronic heart muscle disease of our patients.

Perhaps it will be argued that cases of chronic heart muscle disease appear to live longer or develop decompensation more slowly after removal of foci of infection. So far as I know no well-controlled series of cases has ever been published on this subject. I have not been impressed with

any beneficial results definitely and exclusively attributable to removal of foci of infection in cases which have come under my own observation. Cases of hypertension heart discovered early may live for a number of years in comparative comfort and free from severe decompensation if given a regime tending to reduce activity of various kinds. A reduction in blood pressure will tend to longer expectancy. The factors of reduced activity, reduced blood pressure, and degree of myocardial exhaustion, must all be evaluated carefully when discussing the effects of removal of foci of infection upon the expectancy and rate of development of decompensation in hypertension hearts and in coronary sclerosis. As every one knows, the determination of the degree of myocardial damage is a difficult problem. Under these circumstances it is at present impossible to say accurately how much good is really done by removal of foci of infection and the argument "exjuvantibus" can carry little weight in a discussion of foci of infection as a factor in the etiology of chronic heart muscle disease.

THE STUDENT HABIT

When the modern student compares his outfit of brain with that which was given to his ancestor in remote times he meets with another surprise. The men who lived in Europe twenty thousand years ago were just as well equipped as he is as far as concerns size and form of brain. What did those ancient hunters do with so big a brain? They had no professional examinations to pass, no briefs to master, no leaders to write, no mathematical problems to solve, no ancient classics to translate, no sermons to prepare and preach. If brains were given to us merely for such purposes, then those ancient hunters had somehow come by a superfluity. Brains, however, serve the needs of much more than the intellectual side of our lives; beneath the intellectual centres lie a myriad of others which subserve more menial duties—by the exercise of which we fill the cup of life's enjoyment.—*Sir Arthur Keith, Lancet, Oct. 3, 1925.*

HONORIFICABILITUDINITY R. I. P.

Serious case of alphabetical elephantiasis reported in the *Daily Telegraph:*

At the recent Dental Exhibition attention was drawn to a tremendously long word used to describe a germicide. Long as it was, it is easily beaten by one that appears in the program of the London Medical Exhibition, opened at Westminster yesterday. It is as follows: "Dimethylaminobenzoyldimethylethylcarbinol." The substance is stated to be a hydrochloride, used as a spinal anesthetic.

Dr. Johnson outdone by some Cockney ward-walker!—*The Living Age.*

HEART PAIN: VARIOUS TYPES AND CLINICAL DIFFERENTIATION*

CHARLES N. HENSEL, M.D.
St. Paul

A cursory review of the standard medical text books fails to show any classification of heart disease based on the symptom, pain. Clinically, it is often imperative to make a differential diagnosis based largely on this symptom. To facilitate matters I have found it helpful to divide heart pain into the following groups: pain of angina pectoris, pain of coronary infection, the pain of aortitis, pain of myocardial origin and pain of pericardial origin, and will discuss each type in detail.

Pain of Angina Pectoris.—The pain of angina pectoris arises in the root of the aorta, just above the aortic valves, may or may not be associated with coronary narrowing and is felt by the patient as a seizure of severe pain located high in the sternum. In this position it may remain fixed or may radiate into the left axilla and down the left arm to the hand and fingers, or up into the shoulder, neck or jaw. Occasionally, the pain may radiate down the sternum onto the abdomen, producing muscle spasm and skin hyperesthesia in the epigastrium and along the right costal margin, thus suggesting gallbladder origin.

Angina usually occurs in the fifth or sixth decades of life and is supposed to be chiefly a syndrome of city life. Men are especially susceptible, particularly those whose occupation causes stress, strain, anxiety, worry and great responsibility. Therefore, we find doctors, lawyers, bankers, stock brokers, etc., making up the majority of its victims.

Wenckebach speaks of the great increase in the number of cases seen by him in the troublous and uncertain times in Austria after the war, recalling upwards of a thousand cases where previously his entire years of practice had yielded not more than one hundred and fifty cases.

In a typical case, the first seizure may occur in a robust, vigorous man of about fifty years who has previously considered himself healthy. One day while running for a street car, or hurrying up a flight of stairs, or engaging in some unusual exertion as on a hunting trip, he is suddenly seized

*Read before the Southern Minnesota Medical Association, Owatonna, Minnesota, May 18, 1925.

by this sternal pain and believes he is going to die. He stands transfixed and staring apparently not daring even to breathe for fear of increasing the agony. It is surprising how long a patient may go without seeming to breathe during an attack.

In most heart conditions dyspnea is an important symptom; in angina pectoris pain is the dominant symptom. During an attack the heart rate does not alter nor is the rhythm disturbed. Beyond a slight increase in hardness the pulse shows likewise no change, nor are the heart tones usually affected. The heart may show slight enlargement and the sounds show a slight shortening of the apical first or even a faint soft systolic murmur confined to the apex.

The blood pressure during an attack may be unaffected, or slightly or moderately elevated, many patients showing systolic readings ranging from 160 to 200 mm. during the attack with a drop to the previous level as soon as the attack has subsided.

The attack may last one-half to two, three or five minutes. After it has passed the patient may feel a little shaken, but soon is able to proceed as usual until the next seizure. Death may occur in the first, second, or third attack, or there may be daily attacks for years.

Once these seizures are established the victim soon learns that while he may be fairly active in the morning he must be more and more cautious to avoid attacks as the day advances. He is particularly susceptible in the evening shortly after dinner, when exertion may precipitate an attack, or, if the meal has been a hearty one, he may even sustain an attack while sitting quietly in a chair reading the paper.

While exertion is the chief exciting cause, excitement is a close second, and as the explosive pathways become better traveled, such minor things as swallowing, coughing, sneezing, straining at stool, etc., may bring on an attack.

During the free intervals one is often surprised to find so little apparently wrong with the heart. Allbutt has said that many of these angina sufferers show so few signs between attacks that if they submitted to a life insurance examination and said nothing they would very likely be passed. It is the robust, slightly overweight individuals who tend to suffer from angina and not the thin, feeble, debilitated individuals. Furthermore, in a majority of instances the heart rhythm is undisturbed and the rate unaltered either during the attack or in the free interval.

Syphilis plays a rôle of minor importance, as shown in a group study by Levine,* in which he found only 7 per cent with positive Wassermann or other positive signs of syphilis. An equal percentage showed signs of diabetes.

Perhaps the most striking finding in this study of Levine's was the incidence of recent coronary infarction in the autopsies of cases with a history of angina. A majority of these showed blocking by thrombus or clot in one or more twigs or prominent branches of the coronaries. The natural deduction is that patients with angina pectoris live until a sufficiently important coronary branch is blocked. One can readily see how necessary it is therefore to differentiate between the pain of angina pectoris and the pain of coronary obliteration.

Pain of Coronary Infarction.—In a patient the victim of angina pectoris whose attacks have been brief and have passed either spontaneously or with the help of nitrites, one day there occurs an attack of greater severity which lasts from a half to one hour, is not relieved by nitro-glycerine and causes the patient to "lay up" for a few days. Undoubtedly a small twig of the coronary has been plugged by a clot, but collateral circulation has become established and the heart can go on.

Some time later severe pain again occurs which the nitrites do not relieve, the patient is nearly in collapse, with pulse weak, thready, and rapid, face pale and covered with a cold damp sweat which may even drench the chest as well. The heart tones are feeble and of a tic-tac quality, the pain is agonizing and requires morphine in doses of one-half to three-fourths grain, which merely blunts the pain, but does not obliterate it. The patient must go to bed, where, in spite of rest and morphine, the pain may continue for upwards of twelve hours or longer. The blood pressure is low: 80–95 mm. systolic, 50–60 mm. diastolic. The liver may be enlarged and tender, with muscle spasm and rigidity and tenderness in the epigastrium, or without the liver enlargement the pain may be referred down the sternum onto the abdomen, producing muscular rigidity and tenderness suggesting the pain of gallstones. Often these patients vomit, particularly if the attack comes close to a hearty meal (which

*Angina Pectoris. — Samuel A. Levine. Jour. Am. Med. Assn., Sept. 16, 1922.

is frequently the case), and may continue to vomit. Most of them soon show temperature elevation to 100° to 101° with leukocytosis from 15,000 to 30,000. These findings frequently suggest gallstones or an acute surgical abdomen, and some have come to operation and either die on the table or within a few hours after the operation, for the heart is already struggling against an almost overwhelming insult and the slightest additional shock may finish it.

At autopsy these infarcted hearts show leukocytic infiltration in the infarcted area with softening. Rupture resulting in hemopericardium and death or, if the area is not too large, healing by fibrosis and consequent thinning of the wall may be the outcome. The temperature and leukocytosis may be explained on the basis of this inflammatory reaction around the infarcted area in the heart.

In a proportion of these patients a friction rub develops over the precordium which may help in the differential diagnosis. Of course, as shown by Herrick,* a number of these patients with coronary infarction die instantly or within an hour. The rest live twenty-four hours to three or four days, or recover. Many of them present gallbladder symptoms which are very confusing. So difficult is the differential diagnosis that Faulkner, Marble and White,† of Boston, studied the histories of thirty cases of each condition and came to the following conclusions:

(1) That females predominate in the gallbladder group, and males in the coronary group.

(2) That coronary cases average fifty-eight years of age and gallbladder cases ten years younger.

(3) That the gallbladder group gave a much longer history than the coronary group, the former having a history of two and a third years, the latter six and a half months.

(4) That the pain may be confined to the abdomen or thorax in either case. Radiation into the arms favors coronary disease; into the back and under the shoulder blades, gallbladder disease.

Furthermore, coronary occlusion is often associated with a sensation of constriction in the chest never experienced in gallbladder disease. Likewise, the pain in coronary occlusion is likely to be constant while that in gallbladder disease commonly occurs in recurrent attacks of pain.

The authors report some cases, one of which is so dramatic I must include it here:

Case 1. "Superintendent of detectives, aged 60, well developed and obese, weight 220 pounds. Referred by home physician for operation for gallstones. History of repeated attacks of very severe pain in lower right hypochondrium and indigestion between attacks. Never jaundiced. For several years worked hard and no vacations. Following a week's stay in hospital felt better and condition improved markedly. Examination the night before death revealed nothing except heart sounds of tic-tac quality, rate 64, rhythm regular, blood pressure 125/80, lungs clear. No tenderness or spasm in abdomen, liver edge not palpable, urine negative. In view of the extreme obesity and marked improvement after a week's rest in the hospital it was decided that in the absence of emergency indications for surgical intervention he should take a prolonged vacation with dietary restrictions and return in three months. Next day he was found dead on the floor of his room. Necropsy revealed complete arteriosclerotic occlusion of the descending left coronary with infarction in the left ventricular wall, rupture of left ventricle and hemopericardium. Gallbladder contained two gallstones—mucosal lining intact."

E. R. LeCount* of Chicago reported on twenty-six autopsies in which he found coronary occlusion. One on a middle-aged man, the patient of a noted Chicago surgeon, who had been ill for two months with the diagnosis of gallstones. He was found dead in his chair. Autopsy revealed complete coronary obstruction, but no gallstones.

Mrs. Louisa H., aged 65, was admitted to the Miller Hospital in February, 1923, complaining of abdominal pain, nausea and vomiting. She gave a history of frequent typical gallstone attacks. She showed no edema nor jaundice. Temperature was normal, pulse 72 per minute, white blood cells 13,600, urine contained albumin and pus. Blood pressure was not taken. After thirty-six hours operation was performed (no record of cardiac examination). Patient was considered a poor risk on account of obesity, and seven gallstones were simply removed and a drain inserted. Pulse ranged from 120-150 during the operation, and was 90 on being returned to room. Seen at 8 P. M., condition was good. At midnight the patient awoke complaining of pain in the epigastrium, and was dead in one-half hour. Autopsy showed a rupture of the posterior surface of the left ventricle, the pericardial cavity being filled with clotted blood. The coronary arteries were sclerosed throughout, with one area showing a grayish red thrombus adherent

*Herrick: Jour. Am. Med. Assn., Dec. 7, 1912.

†Faulkner, Marble and White: Jour. Am. Med. Assn., Dec. 27, 1924.

*Le Count, E. R.: Jour. Am. Med. Assn., April 6, 1918.

to the arterial wall and occluding the lumen, with a second area of infarction near the site of rupture.

Examples of similar cases might easily be multiplied, but these mentioned are sufficient to show the diagnostic pitfalls and warn us all to be wary in attempting surgery in cases of this type.

Pain of Aortitis. — This type of pain must be mentioned to complete the discussion, but the symptoms and signs of aortitis are different so that confusion need not occur.

We have two types, acute aortitis associated occasionally with influenza, rheumatic fever, or early syphilitic change, and the chronic type in which aneurysmal change predominates and syphilis is the chief etiological factor.

In aortitis the pain is a constant grinding one, high in the sternum, often radiating through to the back, generally associated with systolic murmurs to the right of the sternum, but sometimes with aortic regurgitant murmurs to the left of the sternum. Pain is generally worse at night on lying down or in the daytime on exertion. Dyspnea is commonly present. Usually the pain responds magically to iodides, since the majority are of syphilitic origin.

Pain of Myocardial Origin.—I have described in detail the pain of angina pectoris and that associated with coronary infarction in order to make a clearer differentiation between these conditions and the pain of *myocardial* origin.

While the older teachings were less explicit in separating these as clinical entities, and while Mackenzie still refers to the pains which I class as of myocardial origin, as "toxic angina," it is becoming a gradually accepted fact that these three syndromes have different underlying pathologic bases and physiologic properties.

Just as any skeletal muscle may ache when used too long without rest—or a swimmer may have a painful cramp in the calf muscle, so I believe the heart muscle may respond with painful sensations under certain conditions.

Pain of myocardial origin is, I believe, an expression of heart muscle fatigue or exhaustion. It probably does not occur in perfectly normal hearts, but arises as a result of stress or strain in the presence of systemic toxemia or low grade chronic bacterial infection.

It is not infrequently found in cases of chronic hypertension, particularly in the presence of foci of infection or associated with sudden sharp rises in pressure. It occurs likewise in certain valvular diseases, chiefly in mitral stenosis, where it is a troublesome symptom in a majority of the cases.

Pain becomes increasingly frequent as the mitral orifice grows narrower and of all heart lesions is one of the most crippling to the patient and rebellious to treatment.

Pain occurs also in aortic regurgitation, though far less frequently than in mitral stenosis.

Myocardial fatigue pain is found frequently in women at the menopause, sometimes associated with a soft apical systolic murmur and slight elevation in blood pressure.

I have repeatedly found it in high-strung slightly built women of the asthenic type, between the ages of twenty and thirty years, who harbor some low-grade chronic toxemia, such as recurrent appendicitis or intestinal stasis, and are over-working and under-resting.

It is found often in the debilitated tired mother between thirty to forty years who perhaps has had too many children, has had too much work to do and inadequate rest at night for years, and who has poor posture, ptosis and a flabby abdominal musculature.

The pain of myocardial origin arises in the ventricles of the heart and is referred to the left anterior chest wall, where it is felt usually as a dull continuous ache in the region between the fourth rib, the anterior axillary line, and the lower costal border; the commonest site, however, is in the region over the cardiac apex. If the condition is severe there may be skin and muscle hyperesthesia in this region as well as referred sensations down the left arm. The pain may be increased by exertion or emotion, at times taking on a sharp stabbing character.

But this need not cause it to be confused with angina, for the pain is not in the sternum nor has it that sudden arresting quality of angina nor the magical relief from nitrites; neither is it associated with the signs of collapse, as seen in coronary infarction.

The pain may be continuously present for hours, days or weeks, increasing or decreasing as heart strain increases or decreases. It seems to indicate a temporarily limited heart capacity which may show few signs of decompensation.

It has been my custom for years to try out cases where the condition of the myocardium was not at first determinable with a few tonic doses of digi-

talis. In some cases with pain, the pain has been reduced without any other change in the patient's regime. In others the pain has been increased by digitalis and this called for rest.

A majority of these patients respond to rest and digitalis, which adds evidence to the myocardial origin of these pains. In a certain proportion of cases where the patient is high-strung and the threshold for nervous stimuli is low, nerve sedatives must be added.

Cases of myocardial fatigue pain I believe make up the majority of all cases of heart pain, and when properly diagnosed are remarkably responsive to treatment.

Pain of Pericardial Origin.—This type of heart pain need not be confused with the other types previously mentioned. It is usually of rheumatic origin and consequently comes on earlier in life, being rare after the third decade.

The onset is sudden, associated with fever and usually preceded by joint symptoms. The condition is accompanied by evident signs of cardiac distress, rapid pulse, precordial pain, friction rub, limited diaphragmatic excursion, etc.

Cardiac Asthma. — I cannot bear to close this discussion without referring to that condition called cardiac asthma. It is usually not associated with pain, but the presence of evident cardiac distress has caused it to be confused in the minds of some physicians with angina pectoris.

In great haste in the middle of the night you are hurriedly called to the bedside of a middle-aged man who has been carrying an elevated blood pressure. You find him propped up in bed gasping for breath, fighting for air, lips cyanotic, lungs full of moist râles, pulse feeble and rapid, cold sweat on forehead. He begins to cough up pink to red-tinged froth. You believe he is going to die, so great is his distress.

The cause of this disturbance is that the left ventricle has suddenly given way under the load and the blood dams back into the lungs. This pulmonary stasis is further aggravated by the fact that the right heart is still vigorously pumping blood into the lungs.

Here nitroglycerine dilates the peripheral vessels and relieves momentarily the load on the heart while subcutaneous digitalis tones up the left ventricle again.

In cardiac asthma dyspnea is the predominant symptom, in angina pectoris pain, not dyspnea, dominates the picture.

When angina occurs with dyspnea as well as pain, it means serious myocardial damage and a grave outlook.

EIGHT ETHICAL IDEALS FOR PROFESSIONAL MEN

(1) The professional man should maintain a standard of competency in his own field, entirely apart from all considerations of public opinion or private gain. He may fall far short of his own standard, but he must never blur the distinction between good and poor work.

(2) The professional man should consider his compensation, in whatever form it may be given to him, as a secondary end; and he should not only be content with a modest income, but he should regard it as a part of his professional duty to make it clear to all concerned that a professional man is not interested in amassing wealth.

(3) The professional man should take a personal interest in recruiting for his own and for other professions the most promising of the youth of the nation, and he should take a corresponding interest in the problems of professional education.

(4) The professional man should deliberately cultivate honest criticism of his own and all other professional work, training himself to hate sentimental praise and ignorant faultfinding, and developing to the extent of his ability the difficult part of clear-sighted, constructive, critical judgment.

(5) The professional man should regard himself as a guardian of the public interests in every situation where he finds those interests in jeopardy, because this is his first obligation to the society which has given him his professional education and opportunity.

(6) The professional man should champion complete liberty of thought and expression, whether popular or unpopular, without yielding in the slighest degree his own convictions as to what is true or false, wise or foolish, right or wrong.

(7) The professional man should encourage the experimental spirit in every department of human affairs, because he knows that all human progress has come about by this method.

(8) The professional man should use every opportunity to promote better mutual understanding among men of different classes, creeds, nations, and races, recognizing that his education and experience should enable him to rise above all fears and hatreds, and to serve as an interpreter and peace-maker among men.

—Report of Committee on Ethics of Professional Men's Clubs. Frederick M. Eliot, Chairman.

BARE RADIUM TUBES IN THE TREATMENT OF TUMORS AROUND THE HEAD AND NECK*

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The implanting of bare radium emanation tubes directly into tumor tissue as a therapeutic measure marks a distinct advance in treatment by radiation. Since the use of so-called radium seeds was begun in the Mayo Clinic, about three years ago, approximately 150 patients with malignant tumors around the head and neck have been treated in this manner. Many of these lesions were extensive and fulminating. In many instances the condition appeared hopeless, and treatment was carried out with a view of palliating rather than with the hope of cure. In a number of instances treatment was instituted with the sole idea of trying out the bare tubes, as no other form of treatment was considered worth trying. The results have been extremely gratifying, in many instances a distinct surprise. A number of the patients with advanced malignant lesions have been markedly relieved, and even apparently cured. These results have not previously been duplicated in the clinic with any other form of radium or surgical treatment.

The use of the bare emanation tubes was first suggested by the physicist, Duane, who started working on the problem in Paris in 1908. In 1914, Stevenson, in the Proceedings of the Royal Society of Dublin, reported twenty-two cases in which good results were obtained. Shortly afterward the method was introduced into this country, where it was first used by the late Dr. Janeway in the Memorial Hospital, New York.

Method of treatment. — Small capillary glass tubes containing radium emanation are implanted directly into and around the periphery of the neoplasm. Radium emanation is a gas given off from the radium element and possesses the same radio-active properties as the radium element itself. It loses its activity at a uniform rate of about 15 per cent every twenty-four hours so that by the end of two weeks its action is practically nil. It is thus seen that a tube containing 1 mc. of emanation will, by the time it is exhausted, have given off approximately 132 mc. hours of radiation. The bare tubes or seeds measure approximately 0.3 by 3 mm. (Fig. 1), and are prepared by collecting the radium emanation in a long, capillary glass tube, which is then divided into the proper lengths by means of a gas flame which seals as it cuts. These tubes are embedded in the tissues by means of a long, slender, hollow needle fitted with a stylet, just within the end of which the seeds are placed and then forced into the tissues when the needle has been inserted to the proper depth. The tubes either slough out during the radiation reaction or become embedded in scar tissue. I have never seen them cause any trouble. The tubes can be thoroughly sterilized by carbolizing and the contents are practically sterile, so that no harm results should they become crushed accidentally in introducing. Seeds containing from 0.5 to 1 mc. of the emanation are preferred, as the experiments of Bagg have shown that the tissues in the proximity of each tube are thoroughly radiated by this quantity with a minimum of necrosis. Efficient radiation around such a tube is produced over a radius of about 5 mm., so that in the treatment of a neoplasm an attempt is made to scatter the tubes uniformly, approximately 1 cm. apart. Increasing the quantity of emanation in each tube increases the amount of necrosis but does not appreciably increase the extent or efficiency of the radiation. Formerly tubes containing 5 mc. or more were used, but these have been discontinued, for, owing to the extensive tissue necrosis produced, they tend to gravitate from the area where embedded and may lodge adjacent to large blood vessels or nerves, resulting in severe hemorrhage or unduly painful reactions. Heavy external radiation is given as a routine over the neoplasm and regional lymph nodes just prior to or immediately following the implanting of the bare emanation tubes, in order to lessen the possibility of grafting or metastasis.

ADVANTAGES OF THE METHOD

The embedding of these bare tubes in the tissues has a number of distinct advantages over other forms of radium treatment: (1) the radio-active substance is in much more intimate contact with the tumor than when applied in any other manner and more efficient radiation results with less radia-

*From the Section on Laryngology, Oral and Plastic, Mayo Clinic.

Read before the Southern Minnesota Medical Association, Owatonna, Minnesota, May 18, 1925.

tion of the surrounding tissues; (2) a much higher percentage of the radium rays are utilized than is otherwise possible, for the softer gamma rays, which are ordinarily lost by filtration, as well as the beta rays, are made use of; (3) lesions in areas where it is almost impossible to retain radium applied in any other manner are quite readily and efficiently treated; (4) less trauma is produced in introducing the tubes than by any of the other methods of burying radium, and (5) small areas of recurrence are readily taken care of by means of the seeds.

DISADVANTAGES AND LIMITATIONS OF THE METHOD

There are certain disadvantages in the use of the method which, however, are no greater than with the other methods of radium treatment, except possibly the surface applications, which it far surpasses in efficiency. The danger of hemorrhage must be considered whenever a destructive dose of radium is applied to any neoplasm in the proximity of large vessels, especially tumors of the tongue, floor of the mouth, pharynx, and hypopharynx. The radiation reaction following varies greatly. In an infected field, such as the buccal cavity, it is always quite painful and prolonged, sometimes lasting for several months. It appears to bear a direct relationship to the amount of secondary infection present. A minimal reaction follows the introduction of bare tubes into a subcutaneous tumor through the intact skin under strict asepsis. The puncture wound of the introducing needle heals promptly, sealing off the tumor to prevent infection, and the radiation reaction often is no more severe than that following the application of a heavy but less effective surface treatment. On the other hand, when an ulcerated lesion of the skin or mucous membrane is treated so that the tubes in being introduced pass through an infected area, carrying infection into the tumor, a rather severe and usually quite painful reaction follows. The same is true when the dose is sufficiently heavy to produce sloughing of a lesion on the skin, even though there was no ulceration primarily, as secondary infection invariably occurs. The reaction comes on, as a rule, after approximately two and one-half to three weeks, and lasts for several weeks. Often it takes two or three months for the slough to clear away entirely, especially if tubes containing more than 1 mc. are used. Needlessly severe and painful reactions have undoubtedly followed too intensive treatment, resulting from the desire to play safe in many of these cases. However, I have not encountered any untoward effects which could be definitely ascribed to overtreatment. As further experience permits of more accurate estimation of the dosage for a given neoplasm, the severity of the reaction and the amount of slough will probably be greatly reduced.

PRACTICAL APPLICATION OF THE TREATMENT

Treatment with bare tubes is primarily indicated in certain groups of malignant tumors, but in the clinic it is not being used to the exclusion of surgery in frank surgical cases. The procedure will be considered under three headings: (1) treatment of primary lesion with the bare radium tubes alone; (2) treatment of the primary lesion with surgery, supplemented by the tubes, and (3) treatment of the affected nodes.

Treatment of the primary lesion with bare tubes alone.—This type of treatment has been most effective in: (1) fulminating epitheliomas of the tongue, especially in young persons; (2) recurring, fixed epitheliomas in locations difficult of access surgically without destroying important structures, such as around the external auditory canal and the orbit; (3) lesions of the base of the tongue, oropharynx, nasopharynx, and hypopharynx; (4) recurring malignant growths in which the limits are indefinite; (5) malignant tumors of the nasal fossæ, and (6) malignant lesions when, on account of the patient's age or general condition, surgery is contraindicated.

Fulminating epithelioma of the tongue in young persons often strongly resembles sarcoma, clinically. It grows rapidly, is prone to recur locally and metastasize actively following almost any type of surgical treatment. Brilliant results have been obtained in certain of these cases by the use of the bare emanation tubes. Radiation is most effective in this rapidly growing type of epithelioma, and this, with the slight trauma produced in embedding the tubes, makes them a most satisfactory therapeutic agent. Especially striking results have been obtained in cases in which, besides the lesions of the tongue, conditions prohibiting surgical intervention were also present. In one instance, a young man, aged thirty, with an epithelioma of the left side of the tongue, infiltrating the adjacent floor of the mouth, which had been activated by repeated applications of caustic, was found to have also

marked myocardial degeneration and cardiac enlargement. Intensive radium treatment was given externally with distance and screening, and following this eight bare emanation tubes were implanted into and around the lesion on the tongue. The patient has now been entirely free from any evidence of carcinoma for a year and a half.

A number of recurring fixed epitheliomas in the mastoid and parotid region with extension inward along the external auditory canal, have been treated by these tubes. Complete surgical removal of such lesions is always difficult, and the proximity of the facial nerve adds to the problem. Facial nerve palsy has not been observed by me following the use of the tubes in this region.* In one case in which the neoplasm had caused complete seventh nerve paralysis, marked regeneration followed the recession of the growth produced by buried tubes. Again, epitheliomas of the skin invading the orbit are difficult to control with any type of treatment. Operation without exenteration of the orbit is, as a rule, of no avail. In one such case, the patient wished to try the emanation tubes in preference to operation, although advised that destruction of the globe would probably result. This patient has now been well for eighteen months and has had no impairment of vision in this eye.

Perhaps the most remarkable results from the use of the emanation seeds have been in the group of malignant lesions involving the base of the tongue and hypopharynx. A number of these were active, recurring lesions, whose limits could not be ascertained definitely. They were obviously not surgical, on account of their type, location, and indefinite extent. Radium treatment in this group was quite unsatisfactory prior to the use of bare tubes because of the difficulty of accurately applying the radium over the lesion with any assurance of its remaining there and sufficiently radiating the area without producing too much reaction in the adjacent structures. By means of special instruments I have devised (Fig. 2), tubes are quite readily implanted in the affected region. There is little likelihood that the tubes will become dislodged before the greater part of their radio-active influence has been exerted. One patient had a deeply ulcerated recurring epithelioma, about 1.5 by 2.5 cm., on the base of the tongue, extending onto the lateral wall of the hypopharynx, and a metastatic gland in the neck about 3 by 5 cm. Radium over the base of the tongue and externally had been applied elsewhere. In the clinic, nineteen bare emanation tubes, totaling 13.7 mc., were in-

Fig. 1. Radium seeds with instruments used in introducing them.

*Since writing this paper I have seen one case of facial palsy following the use of radium seeds in a recurring epithelioma just below the floor of the external auditory canal.

serted into the lesion under local anesthesia. Radiation with distance and screening over the cervical region was then given. Immediately after this a block dissection was performed. The treatment was carried out a year ago, and there has been no evidence of recurrence either locally or in the nodes. In treating lesions in this location, one must bear in mind that besides the danger of hemorrhage, there is the possibility of abscess of the lung and pneumonia from aspiration of the slough. One such unfortunate experience followed the treatment of an epithelioma at the base of the tongue in a young man who developed fatal aspiration pneumonia. These dangers, however, are probably no greater following the use of these tubes than following former methods. In fact, more severe hemorrhages from this region followed the use of steel points or surface applications.

Malignant tumors of the nasal fossæ cannot, as a rule, be treated satisfactorily by surgery, and the prognosis is correspondingly questionable on account of their inaccessibility and the difficulty in determining their extent. The bare radium tubes are particularly advantageous in these cases, as they can be accurately placed with very little discomfort, and the effective radiation is fairly well confined to the desired area. A number of intranasal tumors have responded remarkably to this treatment.

The method has been used successfully when advanced age or general condition of the patients with malignant tumors has prohibited operative treatment. Epitheliomas of the mouth, complicated by pulmonary tuberculosis, cancers of the face of patients with serious cardiac disorders, and recently an epithelioma of the lower jaw of a patient with a previously unrecognized pernicious anemia, have been treated in this manner.

Treatment by surgery.—The method has been used to supplement surgery in the treatment of tumors of: (1) questionable operability in the parotid and mastoid region, with invasion of the external auditory canal; (2) extensive, inaccessible malignancies of the buccal cavity, and (3) lesions of indeterminate extent around the face, or in the accessory sinuses.

The method has proved a very useful adjunct to surgery in the treatment of malignant tumors involving the external auditory canal. The tubes can readily be implanted into the portion of the growth within the canal, far beyond the point that could be reached surgically. As a rule, it is preferable to destroy malignant lesions of the buccal cavity by diathermy, or the actual cautery, since this can be effectively accomplished under local and block anesthesia with little discomfort to the patient. However, the tubes are a valuable aid in these cases. After a thorough cauterization the tubes are inserted into any questionable areas, a combination of treatment which has greatly improved the end results. In cases of extensive malignant growths around the tuberosity of the superior maxilla extending into the retromaxillary fossa, the tubes have been used alone, or in combination with the cautery, with good results. The possibility of serious hemorrhage must be kept constantly in mind in dealing with tumors in this region during the period of reaction. Ligation of the external carotid artery may become necessary at any time. Severe bleeding has been encountered in a few of these cases, but routine ligation of the external carotid has not seemed necessary.

Treatment of the affected nodes. — Usually in cases of malignant growths of the mouth and pharynx the local lesion is removed, and, as soon as conditions permit, the regional lymph nodes are

TABLE 1

LOCATIONS OF LESIONS TREATED*

Upper lip	5
Lower lip	9
Cheek	4
Parotid	9
Nose (external)	5
Lower jaw	9
Eyelid	1
Orbit	2
Ear	6
Mastoid	4
Temporal	1
Tongue	22
Base of tongue	4
Lower jaw and cheek	10
Upper jaw and cheek	8
Floor of mouth	3
Buccal surface	6
Palate	6
Nasal fossa	6
Antrum	5
Pharynx, including tonsil	6
Nasopharynx	5
Hypopharynx	3
Metastatic nodes	6

*In most of the cases the lesion involved other areas as well.

dissected out. In the cases reported here, however, the prognosis appeared so unfavorable that excision of the nodes was postponed in most instances until the primary lesion could be controlled. The same course has been followed in a number of cases in which the lesion of the mouth was destroyed by the actual cautery or diathermy. However, the regional nodes have been radiated intensively, using distance and screening, and implantation of the tubes into palpable metastatic nodes directly through the skin. In a few cases, fixed, metastatic masses have been exposed, the radium has been implanted into the tumor, and the wound closed. The involved nodes are thereby effectively radiated, and since the seeds are implanted so far below the surface that very little radiation reaction develops in the skin itself, further treatment with distance and screening can be safely given over the remaining nodes as a prophylactic measure. The primary results in some of these cases have been quite satisfactory, but that more efficient treatment of the nodes is desirable is evidenced by the fact that a number of patients have died from metastasis after the local lesion had been well for over a year. The nodes were excised in a few cases, but not until metastasis was demonstrable clinically. Sistrunk has shown that deferring excision of the nodes until there is clinical evidence of metastasis, reduces the chances of cure approximately 50 per cent. In view of the excellent results in certain of these cases in the Mayo Clinic, the regional nodes are now being excised as a routine following the implanting of bare tubes into the primary growth. Better end results are anticipated.

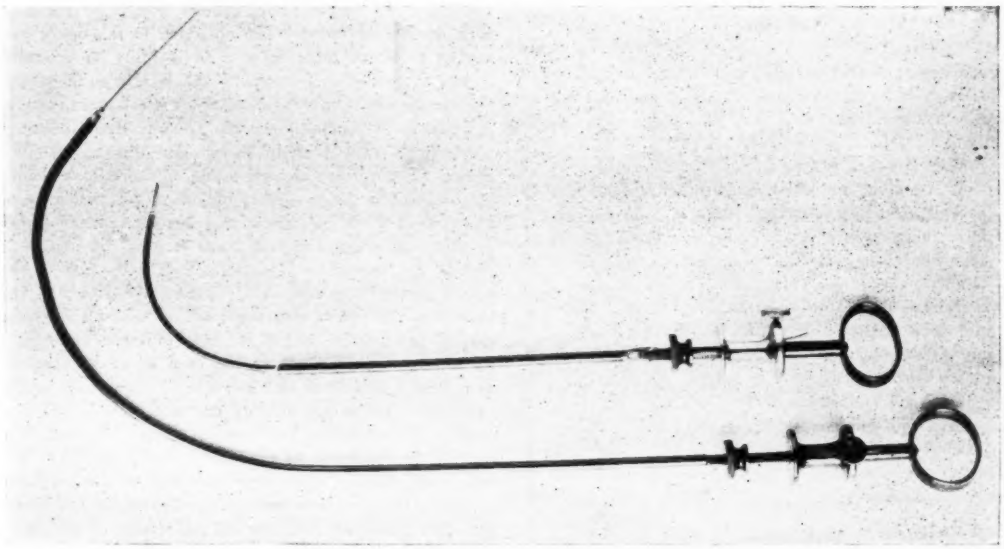

Fig. 2. Instruments used in introducing the seeds into lesions at the base of the tongue and in the larynx.

SUMMARY

The bare radium emanation tubes have effected remarkable primary results. They are of value when used alone in the treatment of the local lesion, following the surgical treatment of the local

TABLE 2

TYPE OF LESION

Basal-cell epithelioma	15
Squamous-cell epithelioma (not graded)	4
Squamous-cell epithelioma 1	4
Squamous-cell epithelioma 2	32
Squamous-cell epithelioma 3	27
Squamous-cell epithelioma 4	13
Basal-cell and squamous cell epithelioma	3
Epithelioma (clinically)	17
Carcinoma	11
Lymphosarcoma	2
Melanoepithelioma	1
Fibrosarcoma	1
Mixed-cell sarcoma	1
Cystadenoma	1
Adamantinoma	2
Giant-cell tumor	1

lesion, and in controlling the metastasis. In all of the cases in which the bare radium tubes alone were used, there was some contraindication to surgery. When used in conjunction with surgery, better results have been obtained than by surgery alone. The patients in this group cannot be considered cured, since none has yet been well more than two and one-half years, but it is quite probable that most of those who have now been free from recurrence for a year or more will remain well.

TABLE 3

RESULTS IN CASES OF MALIGNANT TUMORS OF THE TONGUE

	CASES
Tongue lesions treated over ten months ago	18
Patients not heard from	4
Patients heard from	14
Well	8
Well for two years	1
Well for one and one-half years	4
Well for ten months	3
Recurrence locally	1
Recurrence in glands	2
Dead	3
Dead without local recurrence	
(one and one-half years)	1
(seven months)	1
Type of tongue lesions	
Squamous-cell epithelioma 2	6
Squamous-cell epithelioma 3	8
Squamous-cell epithelioma 4	2
Epithelioma, clinical	1
Adenocarcinoma 3	1

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DISCUSSION ON DR. FIGI'S PAPER

DR. HARRY P. RITCHIE, St. Paul, Minn.: In accepting the invitation to discuss this valuable paper on the particular form of treatment of cancer by the use of radium seeds I fear that I may do so under false pretenses, as my actual experience is limited.

There are very few men in the country who have access to this agent in this form, but it meets with my approval because it permits of surgical judgment in its use in that we may place these seeds exactly at the point of growth and its extensions. We must remember, however, that this is only one form in the treatment of cancer. Generally considered, there are only two ways of treating cancer: one by excision of the growth and the other by destruction in position. The latter includes a great number of agents—radium, *x*-ray, cancer paste, cautery, electrical treatments in a number of forms. The objection to this latter plan applies to all of these agents: an inability to determine results except by inspection. There is always the danger that the growth is only inhibited and not actually destroyed.

Excision, of course, is the positive way if the cancer is widely removed. It seems to me that this has the best chance for a permanent cure. The objection to this form of treatment is the possibility of loss of function or a deformity which will be extreme and therefore carries a great penalty. In surgical attack there is also the chance of making an incomplete removal and, of course, it is well known that such results only stimulate the growth. So I believe in the future that our program is not the discussion of the relative merits of these different agents, but the careful consideration of the case as to whether the new growth is a local one and whether it can be widely removed. This is the primary consideration. If there is danger of incomplete removal by reason of anatomical location the results of this procedure will leave the patient with some horrible deformity. In other words, limitation of surgery in the future will be more carefully considered and such procedure will not be undertaken unless there is more than a fair chance that the growth can be widely removed. Attempts to destroy in position through the surface application of radium and *x*-ray always appear to me as being entirely empirical. There is no assurance of accomplishment. Radium by far outstrips these other agents because we can collect it in such form that it can be used as a surgical agent. Rightly or wrongly in our efforts and opinion we can place this agent exactly where we decide it will do the most good. There is no question of its efficiency when we see the marvelous results of its action as shown in the case of the gentleman here presented. From the description of the surgical tumor this case was absolutely beyond the field of excision. And yet this case in my hands, and I am sure in others, has been subjected to the attempts of surgical removal.

In our discussion of these we must no longer use the poor results from treatment of one in favor of another, but must study the case from all angles to prove whether it is a local growth, not necessarily an early growth. I think "early" is the wrong term. What we are after is "local." We see these cases with a long history and yet every evidence indicates that the growth is still local. We see cases

of short history and yet present glandular involvement. So what we really want is not early or late, but a local growth. Then, it seems to me, the reasonable hope of a cure is its wide removal. Deciding that the growth is out of bounds or is anatomically so situated as to demand gross surgery, then I believe that we should consider these other agents of destruction with or without surgery.

One must come to some definite conclusion and mine at the present time is this—that the hope of a cure, not a three-year or a five-year, but a cure without qualification, is a local growth widely removed.

THE STUDENT HABIT

Many men who are masters of research, who force secrets from Nature by experiment, who prefer to glean their knowledge at first hand and are, I admit, the rarest and highest form of scholars, often despise the habit I wish to extol—the student habit. There have been, and there are, successful medical men who turn aside from books, who leave their medical papers unopened in their wrappers, who prefer to be guided in thought and action by what their fingers have felt and their eyes seen. If by neglecting the student habit they gain something, they also lose much, and it will go ill with their harvest of knowledge if their successors treat them in the same selfish way as they have done their predecessors.

I do not claim for my ideal student, Clifford Allbutt, that he was a pioneer who opened up great new fields of knowledge, but he attains to my ideal because he checked what he saw and what he suspected against the observations and the theories of the great minds that have paved the highways of medicine. Nature had endowed him richly, but he could never have done what he did nor been what he was unless he had acquired the student habit. I speak as an old student of ordinary ability, to young students born into the same happy estate, and I say that the acquisition of the student habit is one of the most valuable assets that a man or woman can carry into any line of life.—*Sir Arthur Keith, Lancet, Oct. 3, 1925.*

All our organs—our hearts, our lungs, our stomachs, and our brains—have been built to meet, not the daily routine of life, but emergencies which occur only at critical junctures. The heart on an occasion can rise to ten times its usual output; the lungs, if pressed, can do nearly as well. As for the stomach, the less said the better; modern civilization tends to throw an unfair burden on it. But as for the brain, the factor-of-safety law holds good; we have, and our ancestors had, about ten times more than ordinary occasions require; our superfluity was given us for emergency. It is just this emergency ration that the modern scholar has to depend on, and there are few, if any, of us who use this extra allowance to its full capacity. You may study to the utmost limit of your endurance, and by the mere act of study you may rest assured that you will do your brain no injury. Infinitely greater harm is done by misuse and disuse of the brain than by overuse.—*Sir Arthur Keith, Lancet, Oct. 3, 1925.*

URETERAL STONES*

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Ureteral stone is a problem of great importance because, first, it is the cause of painfully distressing symptoms; second, because its early removal prevents damage to the kidney which necessarily follows ureteral obstruction; and, third, because it lends itself to very definite diagnosis and localization. The recent work of Hinman on comparative renal function and the success in manipulation of stones by ureteral catheters have brought the question once more to the fore. Hinman, working on dogs, has shown that the recovery of function in a kidney when obstruction has been removed is always good, provided it has not existed too long. If complete obstruction of a ureter has existed over a period long enough to cause increased function in the kidney on the other side, the removal of the obstruction will not make the injured kidney return to normal functional activity because the hypertrophied kidney will continue to hypersecrete. If, however, the hypertrophied kidney is removed and the burden of the excretion is forced upon the impaired kidney, it will regain much of its lost function. This piece of work has changed our viewpoint on operative procedures in kidney and ureteral surgery and upon the importance which the differential functional test bears to them.

The development of roentgenology was undoubtedly the greatest single factor in rendering ureteral stone intelligently accessible to the surgeon. However, it took years of experience, countless operations and post-mortem examinations to differentiate the shadows of stone from the numerous other causes of radiographic shadows. Fecoliths, appendiceal concretions, calcified glands all give shadows which are similar in many respects to the shadows of ureteral stone. Phleboliths are undoubtedly the most frequent cause for confusion in diagnosis. Their round shape, sharply defined borders, and arrangement in chains along the course of the lower ureter, often prove extremely confusing, and when roentgenology was in its infancy many patients were operated for indefinite pains, because of the presence of these shadows, and no stones were found. At the Roentgen Con-

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gress in 1905 the cause of these shadows was discussed but their etiology was not definitely settled. Calcified ligaments, fecoliths and phleboliths were all suggested as causes but it was not until G. O. Clark in 1908 dissected a prostatic plexus containing 18 phleboliths from a cadaver which previously had given shadows in roentgenograms of the pelvis that the matter was settled. A roentgenogram of the plexus after it had been removed showed the same shadows, while a post-mortem roentgenogram of the cadaver was negative. This proved definitely that phleboliths were the cause of many of these shadows, with the result that surgeons began to demand more than a radiographic shadow as an indication for operation.

The diagnosis and localization of ureteral stone today is very accurate. The history may be quite variable. The pain may be a severe renal colic with typical radiation associated with frequency, dysuria or hematuria, or it may be a dull ache of several years duration. Physical examination reveals but slight or no elevation in temperature, tenderness over the abdomen and in the lumbar region on the affected side. The hammer percussion sign may be present or absent. Microscopic pus and blood are usually found in the urine.

A roentgenogram of the kidneys, ureters and bladder, taken after catharsis, usually shows a suspicious shadow somewhere along the course of the ureter. The commonest locations of the stone shadows are at the bladder wall, the uretero-pelvic juncture and the brim of the bony pelvis.

The appearance of the shadows is quite variable, measuring anywhere from two or three millimeters in diameter to ten or twelve centimeters. They are usually oblong in shape with rounded corners. This is, however, by no means constant as they may be round, triangular or irregular. They are usually more dense than shadows caused by fecoliths, calcified glands and pelvic tumors and less dense than the shadows of phleboliths, which are usually round with sharply defined borders and occur in chains.

Certain stones, however, are permeable to the roentgen ray and therefore do not cast radiographic shadows. Newly formed stones do not cast shadows because of their size and molecular structure. These, however, usually pass spontaneously and give very little trouble. Stones composed of pure cystin, xanthin or uric acid give no radiographic shadows even though they are large. This should always be kept in mind and if a good history for stone is obtained, even in the face of a negative roentgenogram, further study should be made to demonstrate the presence of stone.

Cystoscopy and pyelography are the two methods at our command for accurate diagnosis and exact localization of ureteral stone. A stone at the bladder wall may sometimes be seen protruding from the ureteral orifice or the meatus itself may bulge into the bladder even though the stone cannot be seen. Obstruction to the ureteral catheter is another bit of corroborative evidence though it should be remembered that spasm, stricture and anatomic obstruction will all prevent the passage of catheters so that obstruction alone is not conclusive evidence of stone. If the catheter is passed beyond the stone and a definite feeling of grating obtained it may be considered definitely diagnostic. The method of localization with the lead catheter is being gradually replaced by the more exact method of pyelography. The lead catheter even when stereoscopic plates are used is by no means accurate. Only too frequently the shadow of the lead catheter is superimposed on the shadow in question, which makes it impossible to say if the shadow is intra-urinary. Then too in a dilated ureter it is not uncommon for the shadow of a ureteral stone to be at least one centimeter away from the shadow of the lead catheter, which will again lead to a mistaken interpretation.

A pyelo-ureterogram will definitely determine the intra-urinary nature of a suspicious shadow. Braasch in his book on pyelography points out that the shadow of a ureteral stone will always be completely included in the outline of the ureterogram which is characteristically deformed, either by nodular dilatation at the site of the stone, diffuse dilatation extending up to and including the renal pelvis or by complete obstruction to the media with no outline of the ureter above the original shadow. We have, therefore, a method by which we can definitely diagnose and localize stone in the ureter so that there shall be no uncertainty as to its location at operation. It should be remembered, however, that ureteral stones may change their position in a very short time. A roentgenogram taken the morning of the operation will disclose if the stone has remained in the same place as when the patient was examined cystoscopically.

The best method of approach for removal of a ureteral stone requires considerable experience. However, a fairly good general rule for determining the procedure is that stones measuring more than one centimeter in diameter should be removed by an open operation, while stones measuring less than one centimeter in diameter are at least worthy of an attempt at removal by manipulation with multiple ureteral catheters. There are, however, certain exceptions to this rule. If the patient has passed stones previously from the ureter in question, he has a chance of passing subsequent stones much larger than one centimeter in diameter. Approximately 70% of all patients with ureteral stone at the Mayo Clinic, upon whom manipulation is attempted, are successfully treated by this method. The technique consists in the introduction of as many catheters as possible into the ureter beyond the stone. Three and sometimes four catheters may be introduced. The cystoscope is withdrawn and the catheters are allowed to remain in place for forty-eight hours. At the end of this time novocain and papaverin are injected into the ureter to cause as much relaxation as possible. In cases where the stone is in the bladder wall sacral anesthesia affords the most relaxation. After ten minutes a lubricant of oil is injected. Then the catheters are slowly withdrawn by a twisting motion. Occasionally the stone is brought out in the meshes of the catheters. More commonly it passes within the next forty-eight hours following their removal. If one attempt proves unsuccessful a second manipulation may be tried. If this too is unsuccessful an open operation is advisable. The results following removal either by manipulation or by open operation are excellent.

Stones situated at the bladder wall may be delivered by ureteral meatotomy. This procedure should be carried out under sacral anesthesia. Bumpus has recently described a meatotomy scissors which is undoubtedly the best one yet devised. It is made with a stiff shank and has a LaForte filiform mounted on one blade for directing it into the ureter. Cutting the intra-mural portion of the ureter with this instrument permits the successful manipulation of many stones which otherwise would have to be removed by surgery.

Manipulation is not without its dangers. Occasionally following the removal of the catheters complete obstruction results, due to spasm, with severe colic resulting. The already infected urine may set up a pyelonephritis with high temperature, chills and vomiting. When this occurs the catheter should be replaced for drainage. If this is impossible open operation is indicated.

Where the obstruction to the ureter has existed for so long a time that its function has been greatly impaired and the other kidney shows definite increase in dye excretion with the differential functional test, a nephrectomy should be considered because the kidney which has become accustomed to performing all the work will continue to do so and the impaired kidney will continue on its way to complete atrophy. If the stone can be removed at the same operation without too much difficulty, it should be done. If not, it may be left in the blind ureter and no fears may be entertained from it.

CONCLUSIONS

1. The development of roentgenology was the greatest single factor in rendering ureteral stone intelligently accessible to the surgeon.

2. There are many causes of roentgenographic shadows similar to the ones cast by ureteral stone.

3. The lead catheter cannot be relied upon in the diagnosis of ureteral stone.

4. We have at our command in pyelography a means for definite diagnosis and localization of ureteral stone.

5. Stones measuring less than one centimeter in diameter are worthy of an attempt at removal by manipulation. Stones measuring more than one centimeter in diameter usually require an open operation.

6. Stones located in the bladder wall may sometimes be removed by ureteral meatotomy under sacral anesthesia.

7. Where obstruction to the ureter has existed so long that the function has been markedly impaired and the other kidney is hyperfunctionating, nephrectomy is indicated.

THE DIAGNOSIS OF RENAL TUBERCULOSIS*

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The data reported in this paper were obtained from a study of one hundred histories and patients observed at the University Hospital, University of Minnesota, at the Glen Lake Sanatorium for Tuberculosis, of Hennepin County, and from patients observed and treated at the Nicollet Clinic, of Minneapolis.

The diagnosis of renal tuberculosis may be discussed under six headings: (1) clinical history and findings, (2) urinalysis, (3) urologic study, (4) roentgenographic data, (5) animal inoculation, (6) differential diagnosis.

CLINICAL HISTORY AND FINDINGS

In my experience over 95 per cent of all patients having renal tuberculosis complain primarily of acute cystitis. This is not relieved by treatment and is continuous throughout the disease. One exception to this statement occurs when the ureter becomes occluded by stricture, which may prevent urine from reaching the bladder so that it may never become infected, or, if infected, it may recover following complete ureteral stoppage. In early renal tuberculosis, symptoms of cystitis may be present without demonstrable lesions in the bladder. Cases have been reported wherein no symptoms were observed and wherein no findings were reported in the bladder. In such instances the resistance of the patient against tuberculosis is so great that lesions in the kidney are quickly encapsulated before destruction or suppuration occurs so that the lower urinary tract is never involved.

In a small number of histories, I find *hematuria* mentioned as the first symptom. In some, hematuria is the only symptom during the onset of the disease.

Renal *pain* is rarely, if ever, a primary symptom of renal tuberculosis. When occlusion occurs because of ureteral stricture, pain similar in character to that found with any ureteral obstruction may be noticed. Aching in the loin may be present when perirenal infection occurs. Aching over the sound kidney is thought to be caused by hypertrophy of this kidney which is quickly taking on the work of its disabled mate.

Renal *tumor* which occurs in 3 per cent is not a frequent finding and is infrequently discovered by the patient. It is caused by ureteral obstruction so that retention in the kidney pelvis results.

Palpation. When palpation of a tuberculous kidney is attempted, definite enlargement is infrequently discovered, even when the kidney is larger than normal. Slight difference in size may not be determined. Tenderness on deep palpation, particularly if the perirenal tissue is involved, is a frequent finding. The bladder is usually tender to deep pressure. In the female the ureter can be felt as a distinct cord which is extremely tender. This finding is not noticed in the early cases.

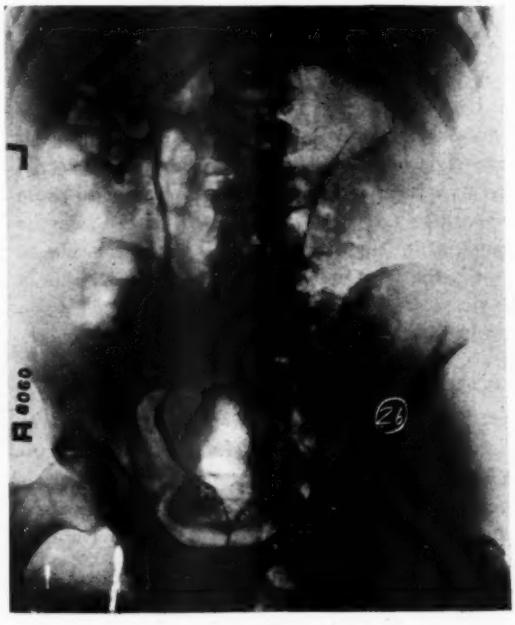

Fig. 1. Injected radiogram of the left kidney which revealed a very early lesion of tuberculosis. This is a cortical abscess which opened into the upper calyx.

Lesions of tuberculosis elsewhere. In my experience 85 per cent of patients with renal tuberculosis have some demonstrable lesion active or healed in the lungs. Tuberculosis of bone is next in frequency and is found alone or associated with pulmonary tuberculosis in 28 per cent. Tuberculous glands are found in 16 per cent. As renal tuberculosis is a blood-borne infection in practically

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every instance, the finding of lesions in the lungs or elsewhere during a general survey should make one think of renal infection as a possibility. A careful study of the urinary tract in the presence of tuberculosis elsewhere will reveal many unsuspected cases.

Lesions of tuberculosis in the lower urinary tract may be the only finding, during a complete physical examination, which indicates the presence of tuberculosis. The examination of the prostate, vesicles, vas and epididymis should always be made a part of general physical survey. A surprising number of patients with tuberculosis of the epididymi and prostate have a renal lesion which is

Fig. 2. Injected radiogram of the opposite kidney, which is normal. Sodium iodide withdrawn from the infected kidney with a syringe. The radiogram reveals retention of the sodium iodide in the cortical abscess.

easily overlooked unless the kidneys are carefully and repeatedly examined. Recently, I observed one patient who had thickening of the left vas deferens without a demonstrable lesion in the epididymis or prostate. Repeated examinations of the kidneys revealed tuberculosis of the left side.

Urinalysis: The characteristic urine in renal tuberculosis is turbid with low specific gravity and contains few other organisms. In the bladder urine, gross blood will be found in 20 per cent and microscopic blood at some time during the disease in 75 per cent. If renal occlusion occurs, pus or blood may never be present. Pus is present in the urine in over 95 per cent of patients. Pus may be present in the urine during one examination and not another.

Tubercle bacilli are found in smears made from bladder urine in 77 per cent, and, if carefully searched for, in 93 per cent of ureteral specimens. One negative smear does not mean that the urine is free from organisms. Large specimens of urine should be centrifuged and several smears made. If secondary infection occurs, tubercle bacilli may be present without being found on smears. I have used the alcohol precipitation process during the last few months, which has made my smear examination easier and has produced more positives. Catheterized urines should be obtained when possible. The smegma bacillus, in my opinion, does not have to be considered if care is taken when the specimen is obtained and if careful and correct counterstaining is done. When large amounts of pus are encountered, the organisms are present in larger numbers. Dr. N. A. Alcock in a recent article finds that both pus and tubercle bacilli occur sporadically in the urine so that he does not cystoscope his patients during the interval when no pus is found. This writer finds that the injection into animals of a specimen of urine obtained from a patient during an interval when pus is not present may not produce tuberculosis. I have watched two patients with bilateral renal tuberculosis for two years. It is quite common to find the urine negative for bacilli and pus at one time and find both present at another. Guinea pig inoculations have been negative two times.

The finding of tubercle bacilli in the urine, in my experience, indicates an active lesion of tuberculosis some place in the urinary tract. This lesion is most often in the kidney. At present a difference of opinion exists concerning the possibility of tubercle organisms passing through the kidney without producing damage or without a lesion being present. My opinion is that clinical evidence does not substantiate the theory that tubercle bacilli can pass through the kidney without producing some lesion. In only one instance did Braasch find a normal kidney following a diagnosis of unilateral renal tuberculosis based on the guinea pig inoculation.

Recently I removed a kidney for tuberculosis which has to date no gross or microscopic evidence of this disease. The entire kidney will be serially sectioned for evidence of minor individual lesions. I have watched this patient for the last three years,

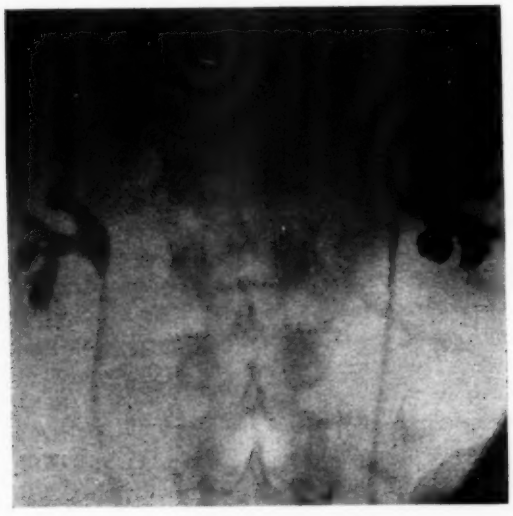

Fig. 3. Injected radiogram. The left kidney contains many cortical abscesses which have broken into the pelvis. The pelvic outline is irregular. On the right side the normal kidney was injected for comparison.

during which time a number of cystoscopies have been done and tubercle organisms have been found repeatedly both by smear and after animal inoculation. This patient has the usual history which is found with renal tuberculosis. She had a pulmonary lesion which is now healed and inactive. The urinary symptoms have not cleared up in spite of her good general condition, so that the infected kidney was removed.

The work of Medlar and Sesano indicates that individual tuberculous lesions of the kidney may heal, although they find this condition rare. They have found lesions in the kidneys of animals which are small and cannot be discovered unless the entire kidney is sectioned. These writers conclude that no evidence is at hand which supports the theory that a normal kidney can excrete tubercle bacilli.

UROLOGIC DATA

The diagnosis of renal tuberculosis is always made from the data obtained during a urologic examination. The history may indicate that this condition is present and the urine may be positive for tubercle bacilli but the urologic study is necessary to locate the lesion and determine its extent.

The majority of patients with renal tuberculosis have a very irritable bladder, which very infrequently permits the passage of a cystoscope without general or caudal anesthesia. Caudal anesthesia has been of great assistance to the urologist, particularly when examining tuberculous bladders. I know that caudal anesthesia has made it possible for me to cystoscope many patients satisfactorily that could not have been done with general or local anesthesia. During caudal anesthesia the bladder reflex is controlled for a sufficient length of time so that cystoscopy can be done painlessly and thoroughly.

Fig. 4. Injected radiogram which reveals a total destruction of the kidney.

There is no other condition in the urinary tract that produces the severe bladder lesions that are found with tuberculosis. One must remember, however, that the patient's resistance may be high and will control the lesions in the kidney so that little

or no bladder symptoms are noticed. A stricture of the ureter may prevent tubercle organisms and pus from infecting the bladder.

An atypical history, together with few urinary findings, may cause one to overlook the diagnosis in spite of careful clinical examination. Braasch has described "silent" renal tuberculosis wherein symptoms usually found are absent and wherein the urological and urinary findings are negative.

The bladder in early tuberculosis may have but slight redness around the infected ureteral meatus with some swelling. The urine from the affected side is usually turbid and comes into the bladder in frequent spurts. As the condition progresses, the inflammation involves the whole bladder and later ulceration begins. The ulcers are angry looking irregular outlined areas situated any place in the bladder. They are not raised and infrequently, if ever, have a raised or depressed edge. They bleed easily and frequently are covered with a very thin slough or membrane. When ulceration occurs, the bladder contracts to small capacity and becomes very tender. The affected meatus dilates and eventually a golf hole appearance is observed.

Tuberculous inflammation and swelling of the bladder may resemble low-lying carcinoma. If the bladder is thoroughly dilated during cystoscopy, the differentiation is easily made. If doubt exists after thorough examination, a specimen may be removed for microscopic study.

Because the bladder contracts and loses its usual shape the location of the ureters and particularly the sound one may be abnormal. When the location of the meati is difficult, I use large intravenous doses of indigo-carmine which may be repeated if necessary. This dye not only colors the urine so that it may be easily seen as it spurts into the bladder, but frequently the mucosa surrounding the opening is stained blue.

Ureteral catheterization is usually easy unless a stricture of the ureter is encountered. This condition which is found in 45 per cent of patients with renal tuberculosis may prevent the passage of the catheter when it is complete, or if partial may require the use of the smallest catheter. When such an obstruction is passed the urine flows rapidly. If the ureter has been occluded for a long period so that the kidney is destroyed, the secretion may be thick pus which cannot pass through a catheter. The urine from a tuberculous kidney flows more rapidly than from the sound side and is turbid and flocculent.

Much has been written and much discussion is made concerning the possibility of carrying infected urine in or on a ureteral catheter into a sound kidney pelvis and thus produce tuberculosis. The constant irrigation of the bladder during cystoscopy, together with the precaution of forcing sterile water or some mild antiseptic through the catheter as it is being passed through the bladder and into the ureter, should prevent this.

Fig. 5. Roentgenogram which reveals typical shadows produced by calcification in advanced tuberculosis of kidney.

To prevent contamination of the ureteral specimen of urine, some urologists fill the ureteral catheter with sterile water, then seal the outer end with a pin before introduction into the ureter. The pin is withdrawn after the catheter passes into the kidney pelvis and about 5-10 c.c. of urine and fluid is allowed to run away before the specimen is collected. Many observers claim false positives from smears and after animal inoculation because of contamination of the catheter during its passage through the bladder. This will not happen if the above precautions are observed. It is possible for

reflux to occur if the valve at the ureteral meatus is incompetent due to ulceration or inflammation. When this occurs, the findings with the ureteral catheter, together with the urinalysis of catheterized specimens, must be evaluated accordingly.

In early tuberculous infection of the kidney, the dye tests are of little or no value. When one or both kidneys have been partially or completely destroyed the dye may be delayed from one or both sides.

From my experience and from the experience of others who have searched carefully for infection in both kidneys, I am of the opinion that bilateral infection occurs very frequently. Some observers hold that in every instance the infection is hematogenous in origin and is primarily bilateral. Some urologists find reflux a frequent cause of infection in the second kidney. When infection occurs through the blood stream, the supposed sound kidney heals after a small primary lesion is present or the lesions that are present may be encapsulated and located so that they do not produce pus or tubercle organisms in the urine. Alcock, who made a careful urologic study of his patients with renal tuberculosis, found bilateral infection present at the time of examination in 47 per cent. In this series I find bilateral infection present in 41 per cent. I am sure that the number of bilateral infections will increase as further data accumulate. Braasch and Scholl found that 34 per cent of their supposed unilateral cases died in one year following nephrectomy. A number of these deaths were undoubtedly caused by infection in the remaining kidney. Medlor and Sesano in their experimental work, find renal tuberculosis bilateral in 88 per cent.

Pyelographic data have assisted me in the diagnosis of renal tuberculosis in a great many instances. In 27 per cent pyelographic data were a factor in arriving at a correct diagnosis. In suspicious cases, I have found pyelographic evidence of tuberculosis several times in which the smears were negative. The deformity may be slight in one minor calyx only or all calyces and the pelvis and ureter may be involved. The early lesions that I have observed are erosions at the tip of the calyx which are irregular and rough in outline. This evidence of cortical necrosis is typical of tuberculosis. As the process continues, the cortical lesions enlarge and the calyces are dilated so that the outline of the pelvis consists of large abscess cavities connected to the pelvis proper by large dilated calyces. The pelvic outline may be entirely destroyed so that only a pyonephrotic sac is seen.

The pyelogram has assisted greatly in following the destructive process that may take place in the kidney after the first small cortical abscess can be visualized. Among the lantern slides which I present today are two which demonstrate that a kidney was completely destroyed in only three months. The first slide shows a small lesion in one calyx. The second one a large pyonephrosis. When patients have other lesions of tuberculosis which may contraindicate operation (unless a kidney is entirely destroyed) or when doubt exists concerning infection in the other kidney, it may be necessary to watch the progress in the kidney that is known to be tubercular. A well filled pyelogram will give one a better idea of the amount of destruction than the amount of pus in the urine or the data obtained from functional tests.

ROENTGENOGRAPHIC DATA

The original roentgenogram will reveal shadows over the infected kidney in 25 per cent of instances. The shadows are typical of tuberculous calcification. They are usually multiple, small and may not cast as distinct a shadow as a renal stone. The shadows are grouped and have an irregular outline with areas wherein the density of the shadows is different. If calcification has been general throughout, a cast of the kidney pelvis may be seen. These shadows are caused by calcium deposit, which is a late stage of healing with tuberculosis and occurs as a late finding.

Animal Inoculation: Animal inoculation furnishes the urologist with a laboratory test for tuberculosis which is practically free from error. The guinea pig reacts much the same as man does to tuberculosis. If tubercle bacilli are injected the animal will develop tuberculosis in six to eight weeks. A healthy stock of pigs should be selected. All animals before being injected should be examined for large glands and other signs of disease. Some laboratory workers test all animals with the ophthalmic test for tuberculosis before they are used. Two pigs should always be injected for controls. Many observers have reported errors in the guinea pig test. If specimens are carefully taken and carefully injected, errors will not occur. When negative pigs are reported in cases which later are found to be positive, the urine which was injected was obtained when the kidney was not

eliminating organisms. Repeated tests should be made when doubt exists. The urine should be injected as soon after it is obtained as possible.

Differential Diagnosis: The acute onset with symptoms of cystitis which is uncontrollable by treatment is typical of renal tuberculosis. Every patient with such a history should be considered a sufferer from renal tuberculosis until this diagnosis is disproved.

Tubercle bacilli may be found in 93 per cent of catheterized ureteral urines so that this organism should be searched for repeatedly. This finding clinches the diagnosis.

The repeated introduction of suspected urine into a guinea pig will reveal tuberculosis if it is present.

Cystitis produced by other organisms may be hard to differentiate. With infections other than tuberculosis the condition responds to treatment in most instances, which is not the case with tuberculosis.

Bladder carcinoma may be confounded with tuberculosis in rare instances. When this occurs a specimen removed for microscopic study will differentiate the two conditions.

When complete stricture of the ureter occurs with a unilateral infection, the diagnosis may be very difficult. The history of a severe cystitis which healed spontaneously together with stricture of the ureter is presumptive evidence of the presence of renal tuberculosis.

The typical shadows produced by calcification and found with healing tuberculosis will be infrequently confused with renal stone.

The pyelogram is typical of tuberculosis, particularly the early small cortical abscess. If healing is in progress when no calcification has occurred, the pyelogram may be hard to differentiate from other conditions.

Renal neoplasm will not cause trouble in differentiation from tuberculosis.

Evidence of tuberculosis elsewhere in the body frequently assists in making a diagnosis. Tuberculosis in the genital tract means renal tuberculosis in the majority of instances.

The resistance of the patient may control this infection so that an atypical history is obtained and the finding may be so unusual that a correct diagnosis cannot be made except after operation.

CONCLUSIONS

1. The diagnosis of renal tuberculosis is not easy and is being overlooked in many instances. This occurs when the patient has good resistance so that the usual symptoms and findings are not present.

2. The first symptoms are those of cystitis.

3. Urologic examination is necessary before a diagnosis can be made.

4. The original roentgen ray will assist in the diagnosis in 25 per cent.

5. The pyelogram may show definite evidence of pathology when the smear is negative and is a factor in the diagnosis in 27 per cent.

6. Bilateral infection occurs in at least 47 per cent of patients infected.

7. Finding tubercle bacilli in the smears clinches the diagnosis.

8. When doubt exists, inject the urine into animals repeatedly.

9. Renal tuberculosis is secondary to tuberculosis elsewhere in the body and should be considered a local manifestation of a general disease.

10. Renal tuberculosis may be differentiated from other lesions of the urinary tract by finding tubercle bacilli in urine in 93 per cent, by *x*-ray evidence in 25 per cent and pyelogram in 27 per cent. Tissue examination will be positive if carcinoma is present. Evidence of tuberculosis elsewhere in the body is present with renal tuberculosis in 90 per cent.

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DISCUSSION ON PAPERS OF DRS. CULLIGAN AND THOMAS

DR. FRANKLIN WRIGHT, Minneapolis: When I was asked to discuss this subject I did not understand that I was to discuss the group of papers, but thought it was to be only that of Dr. Braasch. I did not hear the first paper and heard only a part of the second. Dr. Thomas' paper covers the condition very thoroughly, but there are some discrepancies. He states that the earliest symptom of tuberculosis of kidney is an acute cystitis. That is not true. Tuberculosis of the kidney is essentially chronic. The earliest symptom is an irritable bladder, not cystitis. Another contradictory statement was that if a man has tuberculous epididymitis he may have tuberculosis of the

kidney and this should be investigated. A little further on he says that a man's kidney should not be investigated on suspicion. If he has a tuberculous epididymitis, with no symptoms from the bladder, investigation of his kidney would be on suspicion.

Regarding the antiseptics I have very few words to say. The majority of new antiseptics that are introduced to the medical profession are not introduced by the profession, but by laboratory workers, working for pharmaceutical houses which want something to sell. They hunt around for something that differs a little from something that is already in use. They try it on an animal and find it does not kill it, and then they send it wholesale to the profession throughout the country. For some reason or another the profession is afraid to tell these people that the preparations are no better than those in use, and so we have the reports we have heard this afternoon. The man who first wrote about acriflavine a year later was saying the same things about something else. On his report the manufacturers have deluged the profession with acriflavine and it is being used all over the country.

Now, regarding the use of urotropin. The first thing is that urotropin has, I believe, a definite, fixed place and is useful. The other thing is that it also has a definite toxic effect, which is manifested clearly and should be known by everybody who has used it. Some patients cannot take urotropin because it irritates their bladder. That is a local effect, not toxic. When a patient becomes toxic on urotropin this is manifested by the urine becoming smoky. If it is not stopped at that time the patient will go on and develop a definite hematuria. If it is allowed to go on he will pass degenerated blood, and if allowed to go on still further he will die and the pelves of the kidneys will be found to be filled with this dark brown degenerated blood. How do I know this? When it first came out I had this experience and when the patient died we had a postmortem.

One other thing, Dr. Braasch spoke about the difficulty of getting acid urine. The urotropin does not give off its formaldehyde unless the urine is acid. Very frequently, no matter what you use, it seems impossible to get it acid. In these cases I depend on the mineral acids. I believe dilute nitrohydrochloric acid will always make urine acid. It should be given 12 to 20 drops well diluted in water, three times a day.

Dr. Gilbert Thomas, Minneapolis (closing): In preparing a paper of this kind, several things are necessary. First, to stay within the fifteen-minute limit. I think if Dr. Wright will take time to read my paper he will find that we agree on most things. I do not hope to live long enough not to make errors.

I think Dr. Foley's paper should be discussed. Anyone who sees a great number of kidney cases will appreciate that the anomalies of the upper urinary tract are numerous. Some years ago, Dr. Foley gave us a very good paper on "Kidney Anomalies." Today he worked this subject up a little further and has given us another good paper. He did not have time to show all of his slides, which would have permitted him to bring out many important points which were not covered in his paper. I have a few slides that I will show you instead of discussing the paper further. (Presented series of slides.)

FRACTURE OF THE PATELLA*

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Fracture of the patella, while not as common as a great many other fractures, is of sufficient importance to be reviewed occasionally. Outside of textbooks on fractures there have been but few articles on this subject during the past ten years. As to the number of patellar fractures, Roberts and Kelly had 2 per cent in 1,393 fractures of all bones. Speed's fracture work at Cook County Hospital showed 1.67 per cent of patellar fractures. Scannell found fractures of the patella at the Boston City Hospital for forty years to be 1.7 per cent of all fractures treated there; of these 882 fractures only 11 were open or compound. Moorhead reports sixty-one cases, a percentage of 1.2.

Cause.—Most writers stress muscle action as the important cause of strictly transverse fractures of the patella, and claim that a direct blow usually causes an irregular or a comminuted fracture. They also claim that a fracture caused by a direct blow does not result in much separation of the fragments because the fascia is not torn. Cotton says, "The transverse fractures are nearly, if not quite always, the result of muscle action, and most often of that sudden, very powerful muscle action exerted to save oneself from a fall. With the knee bent at say 60 to 90 degrees the patella is held by the ligimentum patella firmly down and against the condyles, while the full pull of the strongest muscle in the body is suddenly exerted at its insertion on the top of the patella, well out of line with the fulcrum, namely, the convexity of the condyles, and the result is a fracture, approximately transverse, at or about the part of the bone resting on the fulcrum. But this is not all, or the most important part; the force continues to act, and the lateral expansions of the quadriceps tendon—in which the breaking patella has started a rent—tear widely out to either side; the whole extensor apparatus is gone, and there is no hindrance left to oppose separation of the patellar fragments."

Scudder says, "The 'tear' fracture will be transverse and clean cut, the 'blow' fracture comminuted and irregular."

Kellogg Speed says, "The greater percentage of all breaks with the customary transverse line of

*Read at the midsummer Range meeting of the St. Louis County Medical Society, August 8, 1925.

fracture in the lower portion are due to indirect violence. . . . With the leg semiflexed the patella lies at the highest point of the condyles of the femur in a position of weakness and non-support on the intercondyloid fossa. Sudden strain from flexion of the leg or contraction of the quadriceps will thus tend to snap the patella, and tear the ligaments. Comminuted, stellate, and some oblique fractures are due to direct violence and are of infinite variety. They may follow simple transverse fractures with a subsequent fall furnishing direct violence to comminute the bone."

Moorhead says, "Muscular violence is generally regarded as the more usual origin and this generally takes the form of a sudden bending or twisting of the knee in an effort to regain balance to prevent falling after tripping or stumbling; or where some strong pushing force continues to overbend the knee with the limb more or less rigid. Direct violence in which the knee-pan is struck or impinges against an object is a less usual source." However, in several of the patients I have seen there was a history of a direct blow on the patella, the fragments were widely separated, and operation showed the fascia torn. Two of my patients were riding together in their auto when it collided with a street car; each sustained a fractured patella caused by the knees being thrown against the instrument board of the auto. The driver's fracture was transverse, was comminuted, the fragments were widely separated, and the fascia torn. His wife's fracture was transverse, was compound, with fascia torn, and a separation of the fragments. Another patient was riding in an auto when it ran off an embankment, tipped over, and pinned him under the car. He had a compound comminuted fracture with separation of the fragments and torn fascia. Another patient jumped from the window on the second floor of a burning building. One knee had a long, deep cut into the joint; the other knee had a badly comminuted fracture of the patella with separation of the fragments and torn fascia. Of the seven cases I have had those having the greatest separation of the fascia were due to direct blows. These fractures caused by a direct blow were all comminuted; but they also had torn fascia and separation of the fragments. I believe many of these fractures are caused by a combination of both blow and muscle action, the powerful quadriceps muscle exerting a strong pull on the fascia at the same time the blow is received.

If the fascia and ligaments are not torn there is very little or no separation of the fragments. The amount of separation depends on the tear of the fascia or tendon of the quadriceps extensor, the bone being a sesamoid bone entirely enclosed in the tendon. Most cases I have seen were injured by a direct blow on the knee. In each there was separation of the fragments, and operation showed a torn fascia. Evidently the quadriceps muscle was under tension and exerted a pull on the patella at the time the blow was received.

Diagnosis. — Diagnosis is usually easy. The fracture is easily felt. There is immediate disability with loss of power of extension. The fragments can be readily palpated and are more or less separated in most cases—often widely separated. The knee becomes badly swollen very quickly. While the patient may be able to stand or even walk backwards, he suffers severe pain and his knee is very tender. *X*-ray will confirm the diagnosis, and show the extent of the fracture.

Prognosis.—A fractured patella is not dangerous to life unless it is compound and becomes infected. Bony union, however, is not as common as is often supposed, whether treated by open operation or not. Probably a large proportion of fractured patellas result in fibrous union regardless of the kind of treatment. But if the parts heal without too much separation, and especially if the fascia is not torn, or, if torn, has been united, the functional result should be excellent. Moorhead says, "Fibrous union is the rule; it may be so close, firm, and tough that in effect it is bony; but true caseous repair is rare." Speed says, "Fibrous union is the rule between bony fragments in most cases not treated by operation and probably in many operated cases, but in the latter instances this union is more stable and has better backing of the strong lateral ligaments, if approximation is performed with a complete understanding of pathology."

Treatment.—The immediate treatment is to immobilize the entire leg with a long posterior splint. If the fracture is compound, separate the skin opening and pour in all the iodine it will hold. Absolute rest in bed, elevation of the leg, and ice bags will help to relieve the pain, stop the bleeding in the joint cavity, and promote absorption of the swelling caused by excessive joint fluid and blood clots. While in some cases with but very little separation of the fragments and apparently no tear of the fascia, closed treatment may give fairly

good results, most fractures of the patella should be treated by open operation unless contraindicated by some constitutional disease, old age, or a very much weakened general condition. Practically every case that is comminuted or in which there is separation of the fragments has torn fascia lying between the fragments which will interfere with union. There is so much swelling that it is usually impossible to approximate the fragments for some time. Without suturing the fascia the result is a weak knee which is often wobbly. One must realize that operation necessitates entering the knee joint, which demands the strictest aseptic technic as well as experience in bone and joint surgery. An infection resulting from an error in technic will be serious and will most probably cause at least a stiff knee, and may result in death. If proper hospital facilities and careful surgeons are not present the case should be transported to a place where they can be obtained. Most of these cases do better by waiting five to ten days before operation and they can be easily moved on a cot or a stretcher. Various incisions are used—curved both up and down, angling, and straight vertical. I prefer the curved incision with convexity upwards, as this allows less pressure on the scar afterwards and gives better access for suture of the torn ligaments on each side of the patella. After opening the joint all blood clots should be gently wiped away, small particles of bone that are entirely loose or are too badly crushed to grow should be removed, and the fragments brought together. One or two deep sutures should be placed in the fascia on each side of the patella to bring the torn and separated edges of the fascia together; this will also bring the separated edges of the patella together. Then use a row of mattress sutures through the fascia and periosteum across the top or anterior surface of the patella, taking the bite a little ways back from the torn edge; follow this with a running stitch to bring the torn edges of the periosteum together, being sure that no torn edge or flap of fascia or periosteum drops down between the fragments. In badly comminuted fractures a suture encircling the entire bone may be necessary. When there is a good deal of tilting of either of the fragments, holes may be drilled in both upper and lower fragment starting one-quarter to one-half an inch from the edge of the fracture on the anterior surface and directed towards the posterior part of the fractured edge so as to come through on the fractured edge near but not quite to the posterior surface. Then catgut is threaded through these holes, tying the fragments together. All sutures should be absorbable. Very few surgeons use wire for this operation. The skin should be closed without drainage, and a long posterior splint applied, preferably a moulded plaster of Paris splint. Daily massage of the leg will help to keep the muscles in good condition; most authorities advise no motion for two to three weeks.

Results.—In three to six months a good functional result should be obtained. A few cases will have some weakness particularly in going up and down stairs, but this should improve with time. Refracture of the patella when it occurs is generally during the first year. E. M. Cornor of London says that the patella is more often refractured than any other bone. The disability period should average ten to sixteen weeks total and eight to twelve weeks partial disability.

Case 1.—Mr. M. A., aged 29, was injured September 4, 1904, by being thrown from a wagon in a run-away. He struck on the hard road, sustaining a simple fracture of the right patella with separation of the fragments. Operation was performed the next day. Blood clots were removed, holes were drilled, angling into the upper and lower fragments and threaded with catgut, bringing the fragments together. The skin was closed without a drain. Result was a good walking leg with good motion. I saw this man several years after his accident and his right leg seemed to be as good as his left.

Case 2.—Mr. D. R., aged 49, was injured December 12, 1912. He was struck on the head by a falling poker which rendered him unconscious. When he came to, he was unable to walk and had severe pain in his left knee. He had a simple fracture of the patella with a separation of the fragments, which was sutured with catgut. His knee is as strong as before the injury, and motion, while not good, is nearly as free as before, he having had very much limited motion, the result of an old septic knee.

Case 3.—Mr. A. S., aged 45, a merchant, was injured July 26, 1923. While driving his auto he collided with a street car and the impact of his knee against the instrument board of his car caused a transverse and comminuted, though not open fracture of the left patella. He was operated on July 28. Operation showed a fracture at the junction of lower and middle third, comminuted, the capsule being badly torn. The joint was full of blood clots, which were cleaned out. A deep catgut suture, placed in the fascia over the anterior surface of the patella, was imbricated, the distal edge, being shorter, was placed under the longer proximal edge and sutured with catgut, using mattress stitches as in a hernia operation, followed by a running stitch for the top layer. The result was perfect motion and no apparent weakness.

Case 4.—Mrs. A. S. (wife of Case No. 3), aged 42, was riding with her husband when he was hurt. She sustained a compound fracture of the left patella. Operation was

performed August 3, eight days after the accident. Inner side of fracture was not much separated, but outer side was separated more than one-half of an inch and the fascia on this side badly torn. A mattress stitch of No. 3 chromic catgut was placed in the fascia outside the patella; the covering of the bone was loose at the edges of the fracture. These edges were imbricated with mattress stitches, followed by a running stitch of catgut. Result—a fibrous union with some stretching of the tissue and some tilting of the lower fragment. She has, however, a good walking leg with good motion.

Comment.—Angling holes drilled through upper and lower fragments would probably have given a better result.

CASE 5.—Mr. R. N., aged 22, was injured August 19, 1923. He was riding in an auto which tipped over, pinning him down in some water in a ditch, where he was held for about two hours. His injured knee was under water and he says he put his finger in the wound, trying to find out what the trouble was. When he was brought to the hospital, we found he had a compound fracture of his right patella, the opening being one inch long and the fracture comminuted. The wound was filled with iodine, worked into the joint as much as possible. No infection followed. Operation was on the twelfth day. The original wound was reopened and enlarged, extending across the knee. The middle piece of bone was entirely loose and was removed. The capsule attached to the upper fragment had a long overhanging edge between the fragments. Blood clots were removed, the fascia sutured on each side of the patella, as well as across the anterior surface, imbricating the torn edges across the anterior surface, and closing without drainage. Flexion to more than a right angle and a good working and walking leg was the result.

CASE 6.—Mr. J. S., aged 52, was injured December 14, 1923, by slipping on some ice. He sustained a simple fracture of the left patella. Operation seven days later showed the outer part of the patellar fragments separated half an inch and the joint cavity full of blood clots. Capsule was not torn as badly as some of the others. His fascia was sutured similarly to the other cases and, like the others, healed without infection. He has good motion and a strong leg.

CASE 7.—Mr. J. D., aged 40, was injured March 8, 1924. He jumped through the window on the second floor of a burning building just ahead of part of the falling wall and was knocked down by falling brick and debris. He had a deep cut into the right knee joint, a badly cut face, and a comminuted fracture of the left patella. The right knee and face were sutured immediately. Ten days later, the right knee being healed, his fractured left patella was sutured. The bone was broken into five pieces and one loose piece, one inch long by a half inch, was removed. The remaining fragments were brought together, deep sutures were passed through the fascia on each side, the covering over the patella sutured both vertically and transversely, and a posterior splint applied. The result was a very good, serviceable leg, with flexion to more than a right angle, and he is working every day at his old work in the planing mill.

MEMORIES OF THE SALPETRIERE*

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St. Paul

My first view of the Salpêtrière, with the bronze statue of Pinel guarding its entrance, stirred in me varied emotions—pleasure at the gratification of a hope long deferred; a feeling of reverence because here the Great in Neurology wrought and achieved so worthily; satisfaction that I trod the ground where The Emancipator of the Insane, Philippe Pinel, struck off their manacles.

In 1793, Pinel, physician of the Bicêtre, went to those in authority in the Reign of Terror and demanded that he be permitted to break the shackles of those insane in the hospital. "Citizen," said one of those to whom he applied, "art thou insane thyself that thou will unchain such animals? I give them over to thee, but I am afraid thou will be the victim of thy presumption." Twenty-five years later, his pupil, Esquirol, thus describes the misery of these unfortunates: "They are treated worse than criminals and are reduced to a condition worse than that of animals. I have seen them naked, covered with rags, and having only straw to protect them against the cold and moisture and the hard stones they lie upon, deprived of air, of water to quench their thirst, of all the necessaries of life. I have seen them in narrow, filthy cells, fastened with chains, in dens in which one would not keep wild beasts." Pinel commenced to break their chains on the very day he was authorized to do so. In less than a week, he freed at the Bicêtre, fifty lunatics —some who had been in chains for ten, twenty and thirty years. Two years later, at the Salpêtrière, he carried out the same reform. For many years these two hospitals were the only ones where this barbarous system was not in force, and for a quarter of a century after Pinel broke the shackles so dramatically at the Bicêtre, his progressive ideas that everything should be done to alleviate the physical and mental sufferings of the insane, to treat them as patients—as human beings, not animals—to show them kindness and gentleness, advanced not one step. Pinel was fortunate; it was the fullness of time; he had a knowledge of insanity, and he was favored by circumstances. From those deplorable times to the non-restraint of today is a

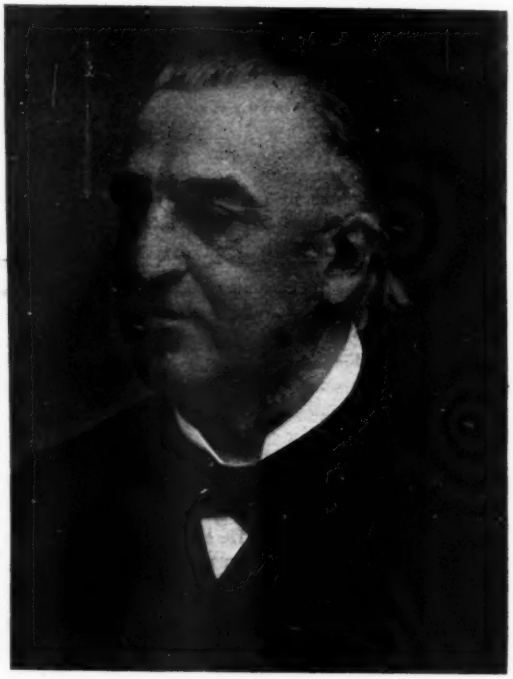
*Read before the Minnesota Neurological Society, September 22nd, 1925.

long cry. Pinel, Pasteur, Charcot—a mighty triumvirate—Frenchmen Three!

In his autobiography, Mark Twain states that he had an excellent memory and that he remembered especially well the things that never happened. In these jottings, the occurrences actually happened.

Shortly after the death of Charcot, through the courtesy of his son, Jean Charcot, I had the opportunity of seeing French neurology at the close of its golden age. Although Raymond, Brissaud, Marie, Feré, Janet, Dejerine, Gilles De La Tourette, and other brilliant workers, were in the heyday of their usefulness and brought great renown to the French School, yet, after Charcot's death, Paris ceased to be the neurological mecca. Professor F. Raymond was genial, personally attractive, wonderfully equipped for his work. His pupils were devoted to him and his audiences cordial and appreciative. To me, Charcot's successor as a lecturer was particularly pleasing—his enthusiasm was inspiring and contagious; he carried one along with him on his current of thought, and there was a feeling of deep regret when the lecture was ended; but his brilliant efforts were in vain. French Neurology produced but one Charcot, and he was dead. Doctors and medical students passed Paris by, and Vienna became the medical mecca.

The Salpêtrière was then, and is now, justly famous—a marvelous reservoir of clinical neurology, inexhaustible and beyond compare. To the Salpêtrière and its distinguished staff, neurology will forever be a debtor. Notable among the cases I saw there was a patient suffering from tabes dorsalis, in whom the radius fractured as the pulse was being counted. While fragility of the bones in paretics and tabetics had long been a matter of anxiety to physicians, I had never seen an instance of such marked fragility. Before the discovery of Erlich, the care of paretics was a constant menace to one's mental equanimity, but since the use of the arsenicals in neurosyphilis, this danger of fracture has been, as far as my experience goes, eliminated. In another tabetic, Charcot twisted the arm around on itself several times. I was unable to understand this medical legerdemain until I observed that the head of the humerus had been entirely absorbed—an extreme illustration of the Charcot joint, which at this time was believed to be due entirely to lues. Now we know that it occurs in syringomyelia, compression and destruction of the spinal cord, severance of the nerves and disease of the peripheral nerves. Rare manifestations of this neuropathic arthropathy are the Charcot spine and involvement of the interphalangeal joints; the cervical and lumbar spine would seem to be the point of selection in the former. Arsphenamine, unfortunately, does not appear to have acted as a deterrent in these joint affections. Patronymics are to be deprecated, yet in these arthropathies, the designation Charcot has been happily applied.



PROFESSOR J. M. CHARCOT.

Another interesting case in this neurological curiosity shop was that of a man who, at two years of age, had an attack of infantile paralysis and thirty-two years later developed progressive muscular atrophy, while from childhood he had suffered from migraine. Charcot believed that poliomyelitis predisposed to progressive muscular atrophy. My experience has not confirmed this opinion. Late epidemics have shown us that poliomyelitis does not confine itself to the anterior horns, but may, to a greater or lesser degree, involve the entire cord. Byrom Bramwell showed us that in progressive muscular atrophy the morbid process is not con-

fined to the nerve cell but affects the cord substance, the neuroglia never escaping even in its mildest forms. Charcot, because of the intrepidity of his scientific spirit, called down on himself severe criticism; his investigations of hypnotism made him most unpopular with his medical confrères—so much so that he was dropped from the medical society, and his name was anathema. Later these same medical men asked the privilege of reinstating him. His contention with the Nancy School regarding hypnotism was very sharp, the latter asserting that every person could be hypnotized, while he maintained that only the hysterical could thus be influenced. Jean Charcot hypnotized a young woman and explained to the late Dr. Alexander Bruce, of Edinburgh, and myself, in detail, the theory and conclusions of The Salpêtrière School. Hysterical paralysis, he said, could be cured by hypnotism, but hysterical palsy would recur, and after a time hypnosis would be a matter of daily routine, as happened in the experience of a member of the staff. Crime, according to him, was never committed under its influence unless the person was a potential criminal. In the laboratory experiment, if the subject were given a paper dagger and told to stab a bystander, if the blow were struck immediately, he would fall to the ground in a hysterical convulsion. One brought up under the amenities and social proprieties never violates them when under hypnosis. He commanded this young woman to kiss me; she stood motionless, her face a flame of scarlet—evidently greatly embarrassed; her embarrassment was no greater than my consternation. The failure of the experiment, he said, illustrated that a regard for the social conventions was stronger than hypnotic influence. This young girl, he said, had been taught by his father and himself to illustrate the phenomena of hypnotism, and that while she had the greatest confidence in them, yet, even under hypnosis, she could not be made to do anything that violated her social or moral code. One woman, whom I had noticed walking around the hospital on her toes, he used as an illustration of the power of suggestion. She was a devout person and spent much time in the chapel, the walls of which were adorned with suitable pictures, among which was an angel about to take to flight; his wings were extended and he was standing on the tips of his toes. Archaic though this conception of hypnotism is in the light of our day, yet it is a passing phase of great interest in the evolution of our present viewpoint. The awe, the glamour, the superstitious fear, which formerly surrounded it, has long since passed. The new psychology postulates the conscious and the subconscious mind; it is to the activities of the latter that we look for the explanation of hypnosis. This later concept may be also a passing phase in the evolution of our final knowledge, since, in a recent lecture, Dr. Bolton tells us that "the myth of the unconscious mind" is an exacerbation of world hysteria. At the time of my visit, the Babinski sign had just been described by him. Dr. Charcot demonstrated it to me and said its significance had yet to be determined.

One of the most interesting lectures I heard at the Salpêtrière was on epilepsy, by Dr. Gilles De La Tourette. He was a consummate actor and he simulated in a most dramatic manner an epileptic attack. I could scarcely convince myself that it was only a depiction and not an actuality. While in Paris, I spent a memorable morning with Feré at the Bicêtre. He was very gracious; it was a clinic on epilepsy—profitable and interesting. Dr. Feré was a big man physically and mentally, and neurology owes him a great debt. He was using borax, and was very enthusiastic over this form of therapy. Borax would again seem to be coming into its own; its combination with bromides and luminal is perhaps the treatment par excellence for this disease.

On my way to the Bicêtre, I passed the Place de la Concorde, and there arose before me the creaking tumbrils, the maddened crowd, the awesome Guillotine and the knitting women that Dickens has immortalized in the days that were red. A most vivid memory is that of a Parisian cocher, a unique personality, devoted to absinthe, with bulbous nose, red face and bleary eyes. Usually I went to the Salpêtrierè by the Seine. On one occasion, when I drove, it was a memorable and never to be forgotten experience; his pour boire was moderate, but his driving was that of a Jehu; his whip was always in motion and suggested the morning of July the 4th; one was in imminent danger of killing or being killed.

It did not take long to discover that French, as I spoke it, was persona non grata; wise forethought had provided me with a letter of introduction from Noyes Brothers and Cutler to their Paris

representative. There was placed at my command a most agreeable young man, whose French was impeccable. We visited the French medical publishing houses and book stores. The desired literature was obtained, and I was fortunate in securing a complete set—dating from the first issue of the Nouvelle Iconographie De La Salpêtrière, a journal founded by Professor Charcot, dear to the hearts of all neurologists the world over. This journal has been one of the pleasures of my professional life. The significant fact about this experience was the conversation of this young Frenchman. Apparently, he had recently come from the annual military maneuvers. All true Frenchmen, he said, gladly accepted military service—a war with Germany was inevitable—only in this way could France be made secure and the indignities of 1870 be blotted out. Any moment, he said, a call to the colors might be made. More ardent patriotism one never sees. His only regret was that of Nathan Hale—that he had but one life to give to his country; and this was twenty-nine years ago.

One incident occurs to me. A Russian who had been bitten by a rabid wolf was brought to the Salpêtrierè for treatment. The patient had been terribly torn and badly mangled; serious infection developed; treatment was successful. At this time the name of Pasteur was on all lips. Although he died in 1895 and was buried with such honors as few men receive, a year prior to my reaching Paris, yet a room of the Pantheon still contained the wreaths that decked his bier. Before his day, nothing was known of the cause of virulent illnesses. Surgery and maternity were handicapped by a paralyzing fear. Forty years later, the work of Pasteur had revolutionized medicine. Microbes, a deadly menace, became, by his splendid genius, agents for the restoration of health and the preservation of life. Recently an election was held in France to determine who was the most distinguished Frenchman. By popular acclaim, the laurel wreath was given, not to Louis XIV or the War Lord of Corsica, but to Louis Pasteur, the son of a tanner, the world's greatest chemist.

Charcot was the Macaulay of French neurology; he was the outstanding neurological figure of his day; his writings were waited for impatiently and read with ever increasing eagerness. To us younger men, he was a beacon light—a dynamic urge. His Lecons Du Mardi, a La Salpêtrierè, were deservedly famous; their publication attained an unprecedented popularity. Charcot revealed to the medical world the inexhaustible storehouse of the Salpêtrière and immortalized both it and himself. His last years were shadowed by the jealousy and acrimonious criticism of distinguished confrères; he suffered from valvular heart disease; age crept apace, yet his dignity and serenity were unruffled and his alertness, mental vision and scientific ardor were undimmed. Thus, in his 68th year, died Professor J. M. Charcot—a fit closing of a great life, the glory of whose setting sun surpassed in splendor its noonday radiance.

Spleen and Red Bone Marrow.—The Council on Pharmacy and Chemistry published a preliminary report of recent work with a mixture of spleen and red bone marrow. At one time desiccated spleen and a preparation of red bone marrow were described in New and Non-official Remedies. Later they were omitted because clinical experience with them had been disappointing. Recently, C. D. Leake and his collaborators have studied the effects of spleen and red bone marrow given separately and in combination. From their studies, these investigators conclude that a combination of spleen and red bone marrow is much more efficient than either spleen or red bone marrow alone. They conclude also that the administration of such a mixture has a beneficial effect on simple anemia, but is without effect on pernicious anemia. While the results do not permit a definite judgment, the Council believes that they are sufficiently favorable to warrant a thorough investigation of the effects produced by this combination on cases of simple anemia. The Council reports that Lehn and Fink, Inc., market Spleen and Bone Marrow Desiccated of declared composition, and that the Wilson Laboratories market a preparation under the proprietary name "Spleenmarrow" stated to be an extract of spleen and red bone marrow, but the method of preparation of which is not disclosed. (Jour. A. M. A., Sept. 5, 1925, p. 744.)

Robinson's Pernicious Anemia Cure.—One W. A. Robinson of Sisseton, South Dakota, has been exploiting an alleged cure for pernicious anemia during the past three or four years. His statements regarding the cause and cure of pernicious anemia prove him to be utterly ignorant of medicine. Robinson charges thirty dollars for a treatment. From letters received from physicians it seemed that the main part of Robinson's "Cure" was coarse sand, and this is confirmed by the report of the A. M. A. Chemical Laboratory. A physician has reported the case of a patient suffering from pernicious anemia who had taken the Robinson treatment and who apparently died from hemorrhages caused by the sand which had been swallowed. (Jour. A. M. A., October 24, 1925, p. 1323.)

DISORDERS OF SPEECH*

SMILEY BLANTON, M.D.
Minneapolis

Speech disorders may be classified from the descriptive standpoint as follows: (1) delayed speech, (2) oral inactivities, (3) letter substitution, and (4) stuttering, which includes stammering.

DELAYED SPEECH

The normal child begins to use voluntary speech when it is about fifteen months old. If speech is delayed a year beyond this time the child should be examined by a physician who is familiar with nervous diseases. The four common causes for delayed speech are: (a) lack of mental development, (b) lack of necessity for speech, (c) continued illness with extreme malnutrition during infancy, and (d) unhealthy emotional attitudes.

Lack of mental development.—The brain must be normal in order that the child develop speech. Sometimes the brain is injured through accident or disease, and speech does not develop. If the injury is discovered early and treatment is instituted, speech may be improved, but if the child is left untreated for several years after the injury, the chances for improving speech are very remote.

Lack of necessity for speech.—Speech is developed in response to definite needs. If the child has too much done for it, speech is delayed or the speech is a jargon which no one but the parents can understand. Such children may be made to talk by refusing to give them what they want until they make an attempt to ask for it.

Continued illness with extreme malnutrition during infancy.—Speech disorders caused by illness or malnutrition should be referred to the physician for treatment. Such cases are not common.

Unhealthy emotional attitudes.—We find children who are extremely negativistic and who refuse to talk, and develop a condition of mutism. Sometimes we find even young children with emotional attitudes that are similar to those which we find in older people, whom we call hysterical. Here, there is a definite emotional conflict which shows itself in a physical symptom, causing the child to refuse to speak. Such cases need careful study and training, and in nearly every such case the home conditions must be modified.

ORAL INACTIVITIES

These consist of a slurring or indistinctness of speech, which may be due to some organic disease of the speech organs, to some emotional conflict, or to some difficulty in the learning process. The learning process is often interfered with through unwise attempts on the part of the parents or teachers to give the child phonetics. Some of the cases of oral inactivity have suffered from malnutrition in early childhood. The treatment consists of re-education of the speech organs through corrective phonetics, care being taken that the child is not made speech-conscious or over-anxious. In nearly every case emotional re-education is also necessary, since there has been built up a series of little fears and feelings of insecurity because of the speech defect.

LETTER SUBSTITUTION

This is usually called lalling or lisping, and is a substitution of one sound for another—such as *th* for *s*, *w* for *r*, *tsh* for *tr*, and *t* for *k*. This defect is often said to be caused by some abnormality of the teeth or palatal arch. Our observations, however, have led us to believe that a minority of these cases are caused by organic abnormality. The majority are caused by emotional conflicts. Many of them are due to a retention of infantile phonetic habits, and many of them are due to a retention of infantile emotional habits. The treatment consists of two parts: (a) emotional re-education, and (b) phonetic re-education. Even in those cases that are the result of organic difficulties there will be certain fears and emotional reactions connected with the speech which must be eliminated before a cure can be effected. In treating this defect the teacher must do three things:

1. He must determine the position of the tongue (or speech organs) in the incorrectly made sound.
2. He must know the position of the tongue (or speech organs) in the correctly made sound.
3. He must be able to determine what over-reaction of the articulatory organs is likely to give the sound desired.

In working with the child there are three points to be noted, which may be roughly stated as follows: (a) unlearning of the wrong position of the articulatory organs, (b) learning the right position of the articulatory organs, and (c) practice until a correct habitual use of the right organs has been obtained. This will mean that the teacher must have a thorough knowledge of phonetics.

*From the Minneapolis Child Guidance Clinic.

*Read before the annual meeting of the Minnesota State Medical Association, Minneapolis, April, 1925.

Straightening the teeth is rarely necessary. Removing the tonsils and widening the palatal arch is scarcely, if ever, necessary. In the treatment care must be taken not to make the child too speech-conscious. Very often the lisper may, through unwise training, begin to stutter.

STUTTERING, WHICH INCLUDES STAMMERING

Stuttering, under which we include stammering, may be described as a break in the rhythm of speech due to a blocking or inhibiting of the muscle co-ordinations. It must not be thought of as a disease but as a symptom of any of a number of underlying conditions.

Stuttering is common enough in children to constitute a very serious difficulty. In a personal survey of six thousand school children of Madison, Wisconsin, it was found that eight out of one thousand stuttered. Dr. Wallin found in a survey of St. Louis school children that seven out of one thousand stuttered. The average percentage of stuttering found in many surveys in this country and abroad shows that about nine children out of every thousand stutter.

Not infrequently we hear it claimed that children who stutter will overcome it in time. In order to determine the number of boys and girls who reach eighteen and still stutter, a personal survey was made of fourteen hundred members of the entering Freshman class at the University of Wisconsin. It was found that one per cent of the students had a marked stutter, and one per cent had a mild stutter, making two per cent in all. It will be seen from these figures that stuttering is not outgrown to any great degree, and even though the defect itself disappears there remains the defect in the emotions—an undue sensitiveness, a feeling of inferiority which interferes with the progress of the individual.

There is, apparently, very little relationship between the severity of the symptom and the severity of the emotional difficulty which is the cause of the symptom. Many people have a very slight defect, so slight that it cannot be noticed, but they feel severely handicapped because, as one boy expressed it to me, he never knew when he was going to have trouble with a word, and, even though he stuttered very rarely, meeting people and adjusting to groups were a terrible strain on him.

Curiously enough, the distribution of stuttering in boys and girls is very disproportionate. There is four to six times as much stuttering among boys as among girls. Just why this is so, no one knows. When a girl does stutter, however, it is just as difficult to overcome the defect as it is in a boy.

We do not find any explanation of the cause of stuttering through the examination of the bodily organs. Of course, such conditions as malnutrition, diseased tonsils, carious teeth, and nasal obstructions may cause an increase in the natural irritability of the nervous system, but those conditions are not the cause of stuttering.

Moreover, stuttering is not inherited. A sensitive nervous system is inherited, on the basis of which stuttering is likely to develop without the proper discipline and training.

Speech is man's chief medium of adjustment to other people; this is the great function of speech. Speech is the chief means by which we come in contact with our fellow beings. It is the way in which we adjust ourselves to the great group. Stuttering, then, is caused by the fear, partly conscious and partly subconscious, of meeting the group. The child fears to meet the group, but he also has a desire to do so. He would like to flee away from the situation altogether. He would like, if possible, to meet the situation. These tendencies to flee away and to meet the situation come into conflict, and we have a compromise in which we have neither good speech nor absence of speech, but broken, inhibited, stuttering speech.

A search of the emotional life of stuttering children always reveals some of these defects—timidity, strong feelings of inferiority, an over-dependence on the parents, and feelings of inadequacy. In some cases we notice a marked rigidity towards life, an unwillingness to change food and sleep habits, an over-suggestibility, a chronic fear of meeting certain groups of people or situations, and a marked sensitiveness.

The essential characteristic of the temperament of the stuttering child is a marked sensitiveness to social situations. This sensitiveness is really a great virtue if properly trained and controlled. In my own experience with stutterers, I have come to feel that they have the most pleasing and delightful personalities of any group of people that I know of. Their quick responses to social situations, their marked sensitiveness, their keenness of perception of social relationships give them an insight and develop in them a type of personality which is pleasing and appealing. Stuttering, then,

should not be thought of as something that is wholly bad. It should be thought of more as a danger signal which indicates that the child requires very careful training in order that he may make use of a sensitive, over-reacting nervous system.

The treatment of stuttering falls under three headings: (a) physical hygiene, (b) mental hygiene, by which we mean emotional re-education, and (c) relaxation and training in muscle co-ordination.

(a) The laws of physical hygiene are well known to parents, so we will not dwell on this phase.

(b) In mental hygiene the most fundamental thing is the changing of the general emotional reactions and the correcting of faulty attitudes which give rise to tension. The child must be trained to work successfully rather than to day-dream, and to get along well with other children. Some outlet must be found for the child's fundamental, social, and biological tendencies and for his necessity for winning some success.

As part of the re-education we include suggestion. This may be given in two ways: (1) Direct suggestion, by showing the child that he can talk when alone and in certain situations, and then assuring him that if he can talk in one situation he can talk in others. (2) Indirect suggestion is given by the teacher's attitude and by the attitude in the home. It often occurs that the indirect suggestion of the attitude in the home overcomes all of the teacher's good work with the child. Whenever the child does well, he should be praised for it and the suggestion given that he will be able to continue to improve until, finally, the speech defect is eliminated altogether. Gradually, as the child improves, he should be made to give talks in front of other people and to place himself in situations where he will have to talk and answer questions.

Parents should absolutely ignore the child's speech defect. They should not ask him to repeat the sentences and should not seem distressed or irritated when the child becomes blocked or inhibited. Even slight changes of expression on the face are noticed by the stuttering child. An example of this is a boy I once treated for a year without any results whatever. His parents moved away, and he remained in the city in order to go to school. During the year that he was away from his parents, his speech defect was completely eliminated. I asked him why he did not get well while he was at home. He said, "I used to feel so badly at home every time I stuttered because my mother would look distressed and unhappy, and this made me so self-conscious and nervous that I could not get well while I was at home."

(c) The use of phonetics or of vocal exercises, such as inflection, change of pitch, breathing, etc., is to be avoided. There is no difficulty in the child's speech mechanism. The difficulty is chiefly psychological. Practice in speech, as a whole, and talking to people are to be encouraged because this is really training the child to meet situations. One of the most helpful things is the teaching of general relaxation. In this the child lies down and relaxes the whole body—feet and legs, thighs, abdomen, chest, neck, fingers, forearm, upper arm, and, finally, the tongue, jaw, and face. When the child is completely relaxed he is asked to repeat a sentence, and, later on, to tell a story and to carry on a conversation; and when he can do this, he is trained to carry this feeling of relaxation with him as he goes about his daily activities.

THYROID PREPARATIONS

Reid Hunt has recently pointed out that dosage with thyroid is largely empiric. The labels on the commercial preparations are as a rule not very elucidating. Dosage expressed in terms of grains of fresh gland is about as rational as reference of the dosage of morphin to the fresh juice of the poppy. The iodin content of thyroid preparations has been made the basis for their pharmacologic evaluation, and the work of Hunt indicates that there is a close parallelism between the physiologic activity of thyroid preparations and their iodin content. So long as the laboratory workers can actually measure the comparative potency with considerable accuracy in relation to iodin content, physicians ought to be eager to grasp this easily determined index as a guide to therapy. There should no longer be justification for prescribing "thyroid tablets" indiscriminately, particularly when it is realized that one "tablet" may contain 2,500 times as much thyroid as another "tablet," the range which is shown to be possible. Very few of the thyroid preparations on the market comply with the U. S. Pharmacopeia Standard. If all physicians were to base the dosage in prescribing thyroid gland on the pharmacopeial product, known as "thyroideum siccum" and to assure themselves that the product which they prescribe contains a definite amount of dried thyroid gland, the present state of confusion would be relieved and thyroid therapy would be placed on a more rational basis.

(Jour. A. M. A., Sept. 26, 1925, p. 978.)

HERMAN M. JOHNSON, M.D.
Dawson
President of the Minnesota State Medical Association, 1926

MINNESOTA MEDICINE

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EDITORIAL

Medical Propaganda

The term propaganda acquired a certain unsavory flavor during the World War. This was due to its abuse in the spreading of false reports and may be charged to the war hysteria generally manifest. There is no reason, however, why propaganda should not be respectable in every way.

The national organization of the medical profession is carrying on some very valuable activities for the education of the public in medical matters. Hygeia, the lay medical magazine, is a good example. In our own state, addresses on medical topics are given over the radio by members of the profession in the name of the state medical association. The Minnesota Public Health Association is doing valuable educational work as a private institution supported by public contributions but under strict supervision of physicians. This is all professedly propaganda.

There are certain forms of propaganda which physicians are particularly qualified to undertake. They spend their energies in the study of disease in its relation to individual and public health. Other forms of propaganda for the avowed purpose of increasing practice or income should not be condoned.

Much attention has been given of late to the subject of periodical health examinations. Examinations of this sort are of proven value and should be encouraged. We do not believe, however, that it would be proper for the physician either individually or organized to institute any campaign among the laity to further the idea. Such activity would smack too much of commercialism. The idea of what constitutes a health examination might well be sold to the profession at large. Let lay organizations, if they so desire, carry on any propaganda along this line among the laity.

Recently a propaganda scheme has been circulated among members of the profession in the Twin Cities, the main theme of which is the encouragement of people to pay their doctors' bills. Now doctors' bills should be paid just as promptly and just as fully as any other bona fide obligations. Perhaps they are. If so, we are sorry for the merchants. It would seem that the budget of the average family does not allow for such contingencies as accident and sickness. It is notorious that most families live right up to their income and installments on the auto have to be paid before everything else. By the time the hospital is paid with what ready cash can be raised the doctor may consider himself lucky if his bill is paid on a time basis.

The proposal mentioned is said to have originated in Tulsa, Oklahoma. Local physicians were having difficulty in collecting their fees. Some newspaper publicity artists sign up a number of physicians who finance a series of newspaper articles with captions such as "Have you paid for your last baby yet?" The importance of paying the doctor's bill is the theme of each of some thirteen articles.

It is to the credit of both the Hennepin and Ramsey county medical societies that the proposal was overwhelmingly disapproved. Such propaganda by non-medical men is a two-edged sword and may, without very careful censorship, defeat its object.

The conscientious citizen might well take offense. The proposition is, to say the least, undignified and is of too commercial a nature. Ours is a profession and commercial methods in their entirety cannot be adopted. If we are in business let us

go the limit, refer cases on a commission basis and advertise. If our work is more than a mere earning of a livelihood, let us adhere to our self-imposed ethics and preserve our dignity.

Cardiovascular Disease

The differential diagnosis between primary kidney and primary heart disease is often difficult. The part that arterial change plays in the clinical picture often makes the diagnosis most complicated. The general use of blood chemistry determinations and functional renal tests has aided greatly in this differentiation. Notwithstanding such advance, terminal pathological changes in the kidney in many instances cannot be accurately predicted from clinical data.

In former days it was thought that glomerular destruction was the cause of vascular hypertension. The greater the glomerular destruction the higher the blood pressure, and hypertension over 190 systolic must of necessity be nephritic in origin. With the observance of individuals with blood pressure readings over 200 who lived for years without developing signs of nephritis, came the realization that our conception was incorrect.

With the development of pathology an effort was made to classify nephritis from the appearance of the kidney at autopsy. We were told that in acute nephritis the kidney was slightly large and red; in subacute nephritis, large and white; and in chronic nephritis, small, white and granular. Parenchymatous, tubular and interstitial were the terms applied, depending on the predominance of pathological change found.

Realizing the difficulty in correlating clinical and necropsy findings, purely clinical classifications have been proposed. Christian has divided nephritis into acute, subacute, chronic, with or without hypertension and edema. He also includes a subdivision of arteriosclerosis leading to nephritis.

Neither a purely pathological nor a purely clinical classification is satisfactory. The classification of nephritis as described by Volhard and Fahr has proven most satisfactory to many. Under nephrosis is included toxic degenerative kidney disease showing tubular degeneration, cloudy swelling and congestion of the glomeruli. Nephrosis is differentiated from the inflammatory nephritis, acute and chronic. Other subdivisions include the so-called benign sclerotic kidney showing sclerosis of the larger kidney vessels and the malignant sclerosis in which the large and small kidney vessels are sclerosed and glomerular and tubular changes are present.

We have come to realize that hyperpiesia or essential hypertension is a clinical entity the cause of which is not perfectly clear. The article by Dr. E. T. Bell of the University of Minnesota which appears in this number of MINNESOTA MEDICINE is most interesting. Carefully kept clinical and autopsy records form the basis for his views. His figures show that very high blood pressure cases are more likely to be hyperpiesia than nephritis. Nephritis accompanied by a blood pressure over 240 is rare, the usual range being from 150 to 200. The conception that nephritis is always glomerular with secondary changes in the tubules and secondary connective tissue replacement is confirmed by his studies.

Perhaps the most valuable clinical conclusion to be drawn from his work is the fact that only 5 to 10 per cent of essential hypertension patients die of renal insufficiency; the majority of such patients die of cardiac failure or cerebral accident.

While the majority of cases of hyperpiesia show arteriolar sclerosis of the kidney vessels it is a significant fact that all do not. It is easy to understand how the blood pressure must become elevated to maintain circulation when the elasticity of the arterioles is diminished and the lumen narrowed by the sclerotic process. This is simply a matter of physics where the peripheral resistance is increased. The cause of the elevation of pressure in the cases that do not show the arteriolar change still remains unexplained.

It is such correlation of clinical and pathological data that is going to clear up our conception of this clinical group known as cardiovascular disease and Dr. Bell has made a real contribution.

It has been asserted by responsible people in our part of the country that the places of physicians in rural districts are being filled by osteopaths and chiropractors and that the country people are being forced to depend on the irregular practitioner. I canvassed this three years ago by the postcard method, and concluded that the irregulars are mostly in cities. Recently I received the official list of chiropractors of the state. There are 376 located in the state at the present time and of these, 154, or 41 per cent, are in Minneapolis. To go to the other extreme, only thirty out of the total number are in towns of less than 1,000 inhabitants. — *Dean E. P. Lyon, Proc. Ann. Congress Med. Educ., etc.*

OBITUARY

DR. THOMAS ROY MARTIN

Dr. Thomas Roy Martin died after a very short illness, Sunday, November 15, 1925, at the age of forty-three years.

Thomas Roy Martin was born in southern Wisconsin in 1882. He was graduated from the University of Minnesota academic department in 1904 and received his degree from the medical department in 1907. Dr. Martin served an internship of eighteen months at the City and County Hospital, St. Paul, and until 1911 was an assistant in Pathology at the University of Minnesota under Professor Wesbrook.

Dr. Martin went to Duluth in 1911 to assume charge of the laboratory of the State Board of Health. In 1913 he entered the medical department of the Duluth Clinic, specializing in gastrointestinal diseases. He continued in that capacity until his death. In 1913 he married Miss Jessie Edgerton, who, with their three children, Thomas, Ruth and Frank, survives him. He is also survived by his father and mother, of Mantorville, Minn., and a sister, Mrs. Senn, of Waseca.

Dr. Martin was held in universal esteem. He had been president of the St. Louis County Medical Society and was a member of that body, the State Association and the American Medical Association at the time of his death. Quiet, unobtrusive, kindly, rational and extraordinarily judicious, he brought to his patients and his profession an impersonal eagerness that won him a large following. He is deeply mourned by all the profession in Duluth.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

SCOTT-CARVER MEDICAL SOCIETY

At the regular annual meeting of the Scott-Carver Medical Society, held Thursday, Dec. 3, 1925, at New Prague, the following officers were elected for the ensuing year: President, Dr. Fred H. Buck, Shakopee; vice president. Dr. H. A. Schneider, Jordan; secretary, Dr. H. W. Reiter, Shakopee; delegate to the State meeting, Dr. B. H. Simons, Chaska; alternate, Dr. M. B. Hebeisen, Carver.

A very full attendance was registered. The Society was entertained and banqueted by the New Prague Community Hospital Board, to whom, together with Dr. E. E. Novak and Dr. W. F. Maertz of New Prague, much credit is due for the success of the meeting.

Case reports on "Myeloid Aleukemia" and "Gonococcus Meningitis" were presented by Dr. M. B. Hebeisen.

Dr. John P. Schneider, Minneapolis, gave a talk on "The Classification of the Arthritides" and lantern illustrations of the more uncommon lesions of the stomach and esophagus.

STATE ASSOCIATION REDISTRICTED

At the last meeting of the Council the following plan of redistricting suggested by Dr. H. M. Workman was adopted, subject to ratification by the House of Delegates:

District No. 1

W. F. Braasch, M.D., Councilor

Counties — Dodge, Fillmore, Freeborn, Houston, Mower, Olmsted, Steele, Wabasha, Waseca, Winona.

District No. 2

L. Sogge, M.D., Councilor

Counties—Cottonwood, Faribault, Jackson, Martin, Murray, Nobles, Pipestone, Rock, Watonwan.

District No. 3

H. M. Workman, M.D., Councilor

Counties—Brown, Bigstone, Chippewa, Lac qui Parle, Lincoln, Lyon, Pope, Redwood, Stevens, Swift, Yellow Medicine.

District No. 4

F. A. Dodge, M.D., Councilor

Counties—Blue Earth, Carver, Le Sueur, McLeod, Nicollet, Renville, Scott, Sibley.

District No. 5

J. F. Savage, M.D., Councilor

Counties—Chisago, Anoka, Dakota, Goodhue, Isanti, Ramsey, Rice, Washington.

District No. 6

W. H. Condit, M.D., Councilor

Counties — Hennepin, Kandiyohi, Meeker, Sherburne, Stearns, Wright.

District No. 7

J. G. Millspaugh, M.D., Councilor

Counties—Beltrami, Benton, Cass, Clearwater, Crow Wing, Douglas, Hubbard, Mille Lacs, Morrison, Todd, Wadena.

District No. 8

G. S. Wattam, M.D., Councilor

Counties—Becker, Clay, Grant, Kittson, Lake of the Woods, Mahnomen, Marshall, Norman, Ottertail, Pennington, Polk, Red Lake, Roseau, Traverse, Wilkin.

District No. 9

W. A. Coventry, M.D., Councilor

Counties—Aitkin, Carlton, Cook, Itasca, Kanabec, Koochiching, Lake, Pine, St. Louis.

Dr. W. A. Coventry was appointed by the President as the Councilor for the new district.

NICOLLET-LE SUEUR COUNTY MEDICAL SOCIETY

The annual meeting of the Nicollet-Le Sueur County Medical Association was held at Le Sueur December 8, 1925. Banquet at 6:30 P. M. The business meeting and program was held at the Farmers State Bank.

The following officers were elected for the coming year:

President—Dr. B. F. Smith, St. Peter State Hospital, St. Peter, Minn.

Vice President—Dr. H. B. Aitkins, Le Sueur, Minn.
Secretary—Dr. J. W. Daniels, St. Peter, Minn.
Treasurer—Dr. F. P. Strathern, St. Peter, Minn.
Censor—Dr. W. W. Covell, St. Peter, Minn.

The following were added to our membership:

Dr. Geo. H. Freeman, Superintendent St. Peter State Hospital, St. Peter, Minn.

Dr. J. A. McKean, Montgomery, Minn.

Dr. J. F. Norris, St. Peter State Hospital, St. Peter, Minn.

Dr. J. E. LeClerc gave a very interesting paper on "Head Injuries," followed by a very free discussion.

Dr. H. B. Aitkins gave a paper on "Morbidity in Childbirth."

Dr. Geo. H. Freeman, St. Peter, was elected delegate to the House of Delegates for the State Society meeting of 1926 with Dr. Swan Ericson of Le Sueur as his alternate.

It was decided to raise the allotment for the Gorgas Memorial Hospital.

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Dallas, Texas, on April 19, 1926. Applications may be secured from the Secretary, Dr. H. W. Loeb, 1402 South Grand Boulevard, St. Louis, Missouri.

OF GENERAL INTEREST

Dr. F. J. von Bohland of Belle Plaine is spending the winter in California.

Dr. W. James Marquis, formerly of the Mayo Clinic, is now located in Houston, Texas.

Dr. Hilding C. Anderson of Duluth is spending a year in study at Vienna.

Dr. and Mrs. C. Curry Bell of St. Paul are receiving congratulations on the birth of a daughter.

Dr. C. F. Wohlrabe, formerly associated with the Swedish Hospital, Minneapolis, is now practicing medicine in Young America, Minnesota.

Dr. Isaac Abt has been appointed attending physician in children's diseases at St. Luke's Hospital, St. Paul. Dr. Abt assumed his new duties January 1.

Dr. Edward V. Goltz and Dr. Alfred Hoff of St. Paul sailed this month for postgraduate study abroad. They will pursue their studies in Vienna and expect to return in the spring.

Dr. H. W. Grant of the Miller Clinic, St. Paul, is now in Gratz, Austria, where he is doing postgraduate work in diseases of the eye. Dr. Grant recently finished his three years' fellowship with the University of Minnesota.

Dr. W. L. Burnap of Fergus Falls, who recently suffered an injury to his leg as the result of an accident in which he was struck down by a taxicab, is recovering. The accident occurred in Cleveland, where Dr. Burnap was attending the annual meeting of the Radiological Society of North America. He has returned home and expects to resume his practice within a short time.

Dr. Harry P. Ritchie of St. Paul was re-elected secretary of the Western Surgical Association at the annual meeting held in Wichita, Kansas, December 18 and 19, 1925. Other officers of the Association are: President, Dr. Robert C. Coffey, Portland, Oregon; first vice president, Dr. Neil John Maclean, Winnipeg; second vice president, Dr. Vilray P. Blair, St. Louis; treasurer, Dr. Frank Teachenor, Kansas City.

At a reception held at the home of Dr. and Mrs. S. M. White, November 20, 1925, in honor of Dr. Berglund, the new chief of the department of medicine of the medical school, all the past chiefs of this department were present, Dr. Charles Lyman Greene of St. Paul, Dr. L. G. Rowntree of Rochester, as well as Dr. S. M. White, the retiring chief of the department.

The members of the department of medicine as well as of the Administrative Board were guests.

Mr. Frederick Hein, who has been associated with Noyes Brothers and Cutler of St. Paul for the past twenty-three years as manager of the Physicians and Hospital Supplies department, has resigned his position, effective January 1.

Mr. Hein first came from the East immediately following his graduation from college, intending to return East later to finish his medical course and practice medicine, but he was persuaded to accept the position which he has held since that time, thus changing from a professional to a business career. Mr. Hein expects to become established in business on his own account in the near future.

Plans are being made to run a Golf Special to the next meeting of the American Medical Association to be held in Dallas, Texas, in April, 1926. A Golf Special was run in connection with the San Francisco meeting in 1923 under the direct guidance of H. H. Gray of the Southern Pacific Lines, and proved such a success that the Golf Special is to be repeated this year.

The train will be limited to 125 people and those desiring to attend the meeting on this special must make reservations in advance. The train leaves Chicago, Sunday, April 11, at 6 P. M., and stops will be made at Nashville, Biloxi (Miss.), New Orleans, Galveston and San Antonio, arriving at Dallas, Saturday, April 17. The cost, which includes all expenses from Chicago to Dallas and return railroad ticket will be $215. Those interested should communicate with Dr. F. C. Warnshuis, Powers Theater Building, Grand Rapids, Michigan, at once.

CYRUS NORTHROP

Tribute is paid to Cyrus Northrop, president of the University of Minnesota for more than a quarter of a century, by Oscar W. Firkins, professor of comparative literature, in "Cyrus Northrop: A Memoir," which Mr. Firkins was commissioned to write by the board of regents. The book has just been published by the University of Minnesota press.

"Mr. Firkins," says the *Minneapolis Journal*, "has assembled and written a remarkable biography. In its frankness and minuteness it is true to the most modern standards. The little weaknesses and the boyish vanities are revealed quite unsparingly. The book mirrors the nature of Cyrus Northrop, the man."

NEW AND NON-OFFICIAL REMEDIES

The following articles have been accepted by the Council on Pharmacy and Chemistry:

ABBOTT LABORATORIES:

Arsphenamine-D.R.L., 0.3 gm. ampules
Arsphenamine-D.R.L., 0.5 gm. ampules
Neoarsphenamine-D.R.L., 0.15 gm. ampules
Neutral Acriflavine Jelly 1:1000-Abbott

ELI LILLY & COMPANY:

Para-Thor-Mone-Lilly
Para-Thor-Mone-Lilly P-20, 5 c.c.

MERRELL-SOULE COMPANY:

Powdered Whole Lactic Acid Milk-Merrell-Soule

PARKE, DAVIS & COMPANY:

Boro-Chloretone
Ovarian Residue Desiccated-P. D. & Co.
Capsules Ovarian Residue Desiccated-P. D. & Co., 5 grains
Tablets Ovarian Residue Desiccated-P. D. & Co., 5 grains
Ovarian Substance Desiccated-P. D. & Co.
Tablets Ovarian Substance Desiccated-P. D. & Co., 5 grains

SWAN-MYERS COMPANY:

Ampules Dextrose, 50 Per Cent., 20 c.c.-Swan-Myers

Arsphenamine-D.R.L., 0.3 Gm. Ampules.—Each ampule contains arsphenamine D.R.L. (New and Non-official Remedies, 1925, p. 47), 0.3 gm. The Abbott Laboratories, Chicago.

Arsphenamine-D.R.L. 0.5 Gm. Ampules.—Each ampule contains arsphenamine-D.R.L. (New and Non-official Remedies, 1925, p. 47), 0.5 gm. The Abbott Laboratories, Chicago.

Neoarsphenamine-D. R. L. 0.15 Gm. Ampules.—Each ampule contains neoarsphenamine-D.R.L. (New and Non-official Remedies, 1925, p. 49), 0.15 gm. The Abbott Laboratories, Chicago.

Scarlet Fever Streptococcus Antitoxin-Lederle (Refined and Concentrated).—A scarlet fever streptococcus antitoxin (Jour. A. M. A., May 2, 1925, p. 1338) prepared by immunizing horses by the subcutaneous injection of the toxic filtrate obtained by growing the scarlet fever streptococcus in broth; also by injecting cultures of the scarlet fever streptococcus. It is marketed in syringes containing 1 c.c. and in syringes containing 10 c.c. Lederle Antitoxin Laboratories, New York.

Saubermann Radium Emanation Activator, 100,000 Mache Units.—Each apparatus (New and Non-official Remedies, 1925, p. 315) imparts about 36 microcuries (100,000 Mache units) to about 500 c.c. of water daily. Radium Limited, U. S. A., New York. (Jour. A. M. A., Nov. 7, 1925, p. 1487.)

Para-Thor-Mone-Lilly.—Parathyroid Extract-Collip.—A stable, aqueous solution containing the active principle or principles of the parathyroid gland of cattle, having the properties of relieving the symptoms of parathyroid tetany and increasing the calcium content of blood serum. It is standardized by its capacity to increase the blood serum calcium in normal dogs one unit being defined as one one-hundredth of the amount of solution required to cause an increase of 0.005 gm. of calcium in the blood serum of a 20 kilogram dog. Para-Thor-Mone-Lilly relieves the tetany of parathyreodectomized dogs, and by its continued daily administration in small doses further attacks may be prevented. The product is a most potent therapeutic agent and its use may be attended with great danger unless due precautions are taken. It is claimed to be a specific in parathyreopriva and to have relieved acute and chronic tetany following thyroidectomy, so-called idiopathic tetany and infantile tetany. Para-Thor-Mone-Lilly is marketed in 5 c.c. ampules, each c.c. of solution containing 20 units. Eli Lilly & Co., Indianapolis. (Jour. A. M. A., Nov. 14, 1925, p. 1559.)

Neutral Acriflavine Jelly 1:1,000-Abbott.—Neutral Acriflavine-Abbott (New and Non-official Remedies, 1925, p. 134) 0.1 part, dissolved in karaya gum jelly, containing sufficient sodium hydroxide so that the finished product has a pH of from 8.3 to 8.5, to make 100 parts. Abbott Laboratories, Chicago. (Jour. A. M. A., Nov. 28, 1925, p. 1729.)

PROPAGANDA FOR REFORM

Lactic Acid Milk.—New and Non-official Remedies brings out that there is considerable evidence in favor of the therapeutic value of soured milk—particularly of sour milk containing an abundance of living *B. acidophilus.* Whereas the administration of *B. acidophilus* has for its object the implantation of living *B. acidophilus,* there are reports which indicate that the administration of milk sugar may produce the same results through promoting the growth of aciduric bacteria normally present in the intestinal flora. (Jour. A. M. A., Nov. 14, 1925, p. 1578.)

Side-lights on Intravenous Medication.—Intravenous injection involves difficulties of technic, with the possibility of local injuries to the peripheral blood vessels at the seat of operation. It presents dangers of bacterial contamination; the vehicle as well as the drug is immediately foreign to the blood, and other objections have presented themselves. The Council on Pharmacy and Chemistry has taken a decidedly conservative attitude toward the recognition of the scores of products intended for direct intravenous use. The wisdom of this stand has been attested anew by a recent report of Hanzlik and his collaborators, who report that a large variety of substances cause definite and important changes in arterial blood of test animals, accompanied as a rule by disturbances in physiologic functions. (Jour. A. M. A., Nov. 21, 1925, p. 1645.)

Herradora Products and Intravenous Therapy.—In 1923, the Council on Pharmacy and Chemistry found the intravenous preparations of the Scientific Chemical Co. (Marcus Aurelio Herradora, president) not acceptable for New and Non-official Remedies because of unwarranted and unsubstantiated statements in favor of intravenous and intramuscular administration of drugs and because of the complex and indefinite mixtures as represented by these specialties. From an examination of advertising recently received there appears to be every reason to reiterate the Council's conclusion that "the propaganda contained in the advertising matter issued by the Scientific Chemical Company is detrimental to the rational practice of medicine and to the public health." (Jour. A. M. A., Nov. 21, 1925, p. 1660.)

PROCEEDINGS OF THE MINNESOTA ACADEMY OF MEDICINE

Meeting of Oct. 21, 1925

The Minnesota Academy of Medicine held its regular monthly meeting at the Town and Country Club on Wednesday evening, Oct. 21, the meeting having been postponed one week on account of the Interstate Post-graduate Assembly meeting, Oct. 12-16. 1925.

The meeting was called to order by the President, Dr. H. L. Ulrich. There were 31 members and 2 visitors present.

The minutes of the September meeting were read and approved.

The following case reports were given:

1. Dr. A. W. Abbott (Minneapolis) showed an apparatus for holding a drainage tube in any cavity.

2. Dr. E. M. Hammes (St. Paul) reported two cases as follows:

(a) Female, age 30, married, seen in consultation with Dr. J. C. Ferguson, October 13, 1925.

The family and personal history were negative. The family stated that she had been perfectly well. She left her home on October 12, at 8 P. M., to take part in a musicale and to visit a friend. At quarter to one that night the doorbell rang three times. When they opened the door they found the patient lying in front of the door apparently semi-conscious, bleeding from the nose, with a little blood on her stocking and on the porch floor. The family thought she had been struck by an automobile and placed on the porch. However, there was no sand or dirt in her hair or on her clothes, and no evidence of violence or external injury.

Dr. Ferguson saw her at 10 o'clock that morning, when her temperature was 102, pulse 100. The patient was semiconscious and her neck was slightly rigid. I was asked to see her about 8 P. M. that evening. The patient was semiconscious, resistive, and gave one the impression of a catatonic dementia precox. When her arm was lifted she would hold it in that position and gradually drop it. When an attempt was made to open her eyes she would hold them tight. She was definitely resistive. Her neck was rigid. She had a questionable Kernig. All reflexes were normal. There was no evidence of any bruise on the head or on the body. She continually rubbed her forehead with her hand as if she had a severe headache. Her temperature at this time was 102.5, pulse 100, respiration 25, and her general condition was fair.

A lumbar puncture was done and about 35 c.c. of spinal fluid was removed, which was almost pure blood. Following the spinal drainage there was no special change in her condition. At 10 P. M. she died quite suddenly.

Permission was given for a post-mortem of the head only. Between the scalp and the occipital bone was a small diffuse hematoma about 2 inches wide and 4 inches long. On opening the skull there was a diffuse hemorrhagic exudate extending over both hemispheres, with a small blood clot under the left frontal lobe and a large blood clot about the size of a small egg under the right frontal lobe. The convolutions of both frontal lobes were quite macerated. The skull showed a fracture in the occipital region extending about 3 inches on the right side from the saggital suture longitudinally through the occipital bone. On the left side the fracture line extended from the saggital-lambdoid junction through the occipital bone into the foramen magnum. There were several short radiating fracture lines besides, beginning in the same area.

This patient evidently rang her doorbell, fell, and the impact from the fall on the wooden floor was sufficiently marked to produce the occipital skull fracture with the marked contra-coup hemorrhage of both frontal lobes. The hemorrhage evidently was secondary to the fall and not primary, because its location and distribution were such as one would expect in a contra-coup lesion in relation to a blow in the occipital region.

(b) The second case was a male, age 42, seen in consultation with Dr. A. R. Comstock.

The family history was negative except that his mother died of gastric carcinoma. The present history was negative. The present illness began during the last week in June, 1925, with intermittent pains and headache in the left frontal and occipital regions. This pain gradually became constant and increased in severity. About July 15 a diagnosis of sinus infection was made, and the left middle turbinate bone was removed. This gave no relief. By July 30 his left frontal headache was so marked that he had to discontinue work. The following day he was confined to bed. During that week he vomited twice. On July 27 he was examined by Dr. Morrison and Dr. Ahrens of the Nicollet Clinic. All findings were negative except for a definite choked disc, more marked in the left fundus, loss of right abdominal reflexes, and a moderate increased right knee jerk. The spinal fluid was under increased pressure, but normal otherwise.

Because of the choked disc, Dr. Strachauer performed a decompression operation over the left fronto-parietal region. The intracranial pressure was found greatly increased, but no tumor or abscess could be located. The headache subsided and the patient seemed brighter. With the formation of a hernia cerebri a right hemiplegia with aphasia developed.

I first saw him on August 28, 1925, at St. Joseph's Hospital, St. Paul. He had a marked hernia cerebri at the site of the decompression operation. He had no headache and seemed bright mentally. He presented a right hemiplegia with the usual pathologic reflex changes. He was aphasic. On September 5, about 5 P. M., he developed a generalized convulsion. A lumbar puncture was performed and 40 c.c. of clear, normal spinal fluid under increased pressure was removed. The convulsions continued, the patient became comatose and died within 36 hours.

A post-mortem was negative throughout except for a tumor of the left frontal region immediately under the operative field. This was subcortical, about the size of a large walnut, and was found to be a glioma. The foramen of Magendi was patulous and the ventricles were not distended.

The interesting points in this case are the fairly acute onset, the rapid progress, the marked intracranial pressure

with choked discs, which is quite uncommon in frontal brain tumors. The obscure symptoms emphasize the difficulty of a proper cerebral localization in tumors of this region.

DISCUSSION

DR. A. SCHWYZER: Isn't it possible that some of the bleeding had occurred before this patient fell? When I saw the blood spread over the brain surface and no damage to the brain substance and no cause for the fall was known, I was reminded of two cases of spontaneous hemorrhage I happened to come across, which were very interesting. The first case, a young man, 18 or 19 years old, was asked by two friends to go in an automobile, and the three young fellows raced around the country. When he came home he had a terrific headache and suffered from severe dizziness. There was perhaps a trace of rigidity in the neck. We took him to the hospital and did a spinal puncture. A raspberry-colored fluid came out under markedly increased pressure. The patient recovered.

The other case was similar. A lady about 55 years gave some orders to her chauffeur, who answered in an impertinent way. The lady is of quick temperament and became very angry. She suddenly became dizzy and had to be carried to bed, and a terrific headache came on. There was no fever in her case either. I took her to the hospital and made a spinal puncture. The spinal fluid was raspberry-colored and under increased pressure. Subarachnoid bleeding had occurred. Again, as in the other case, spinal puncture relieved the headache promptly and the patient recovered readily.

There was no later trouble in either of these cases.

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3. DR. ARNOLD SCHWYZER (St. Paul) reported a case of branchiogenetic cyst and showed *x*-ray plates.

History: When the girl, who is now 13 years old, was born, a walnut-sized soft and painless swelling was noticed low down on the right side of the neck. It gradually grew to the size of a goose egg.

In April, 1924, when the skin over it became thin and bluish, it was opened by the family doctor, who mentioned that the contents were a pus-like fluid. From the beginning a large pulsating vessel was noticed along the median side of this mass.

Since the cyst was opened it has drained a watery and partly purulent liquid constantly. There is no pain. The swelling inward and downward from the fistulous opening persisted. It is somewhat emptied, especially on pressure, and then fills out again.

Physical findings: Soft prominence to the outer side of the tortuous, very visibly pulsating large artery, which is the common carotid and runs near the midline at the level of the larynx to recede laterally again along the upper border of the swelling, which is soft and fluctuating. An irregular scar and tiny fistulous opening exists at the upper outer edge of this fluctuating mass. This pus can be expressed.

Remainder of the examination negative. A stereoscopic roentgenogram, after moderately filling the sac with bismuth paste, shows it to reach about an inch below the lower border of the head of the clavicle. The inner two-thirds of the clavicle itself are bulged out over the mass and join somewhat at an angle with the outer third. This by itself proves the early appearance and considerable size of the cyst.

Operation on October 5, 1925, allowed us to peel the sac out of the depth without much difficulty, while at the site of the fistula it was intimately grown to a large vein. The size of the sac was that of a large hen's egg.

Microscopically the lining of the sac was a pavement cell epithelium of from 15 to 20 cells thickness. The outer layers have a tendency to keratinization. The stroma underneath shows a shallow layer of dense round cells perhaps due to the suppuration; and underneath this layer we see unstriated muscle. The cyst is thus branchiogenetic.

Whether or not the cervical sinus itself could not form cysts is a matter of speculation. The marked downward growth of the third branchial arch causes overlapping over the fourth and causes the cervical sinuses laterally. From the third gill cleft the lower parathyroids originate and also the thymus. Kürsteiner under Langhans made serial sections of this area of the necks of newborn children and found that the thymus is liable to lose some of its tissue in its downward growth, leaving, besides the thymus cord, small heaps of the thymus tissue, at times forming little cysts. However, nothing is known that such cysts should become of macroscopic or even surgical importance. The branchiogenetic cysts come practically always from the second cleft.

The patient left the hospital a week after the operation with the wound apparently well healed.

DISCUSSION

DR. MANN: This is an extremely interesting case and reminds us of others which we have seen and some of which we have operated. The last statement of the doctor, that because it was lined with thin or flat epithelium it could not be one of the cysts in connection with the thymus, is, of course, true. The contents of a cyst is more fluid and mucoid when it arises from the inner portion of the branchial clefts and the lining of the cysts is more like mucous membrane with flattish or sometimes cuboidal mucous cells. In rare cases they may be more columnar in type. When they arise from the outer portions of the cleft, nearer the skin, they are lined with flattened cells more of the squamous type, and then the contents show more and more sebaceous material as it approaches the outer or skin ending of the cleft.

The embryonic remnants of the branchial clefts fairly often persist as cord-like or tube-like remnants. When they persist throughout we have a fistula leading from the esophagus high in the neck to its exit much lower in the neck, due to the great downward growth of the external portions of the branchial arches.

When portions of the tubular remnants remain imbedded in the neck, they form cysts whenever the activity of their lining cells produces secretions sufficient to distend their lumens. These clefts are always interesting. The neck portion has grown down a good deal faster than the inner portion so that they slant from the outside upwards and inwards and go a good deal higher than we would expect. The drum of the ear closes the first cleft. No cyst, so far

as I know, has been reported in the first cleft. Most of these arise from the second or third. The only way to distinguish them after you find them is to see whether the line of the small tube-like projection which goes up to the esophagus, when it runs that far, goes above or below the styloid process and the stylohyoid ligament. In dissecting them out, we never know, until we go high into the neck, whether it is from the second or the third. It has to be followed up to that point before it can be distinguished.

I think the Doctor's method was very ingenious; to turn in the stump and thrust it through into the esophagus at a new point, seems to me a very admirable way of doing when the inner portion is cordlike and cannot be treated in the standard method, which is to turn the stump inside out by putting it through its own lumen into the esophagus.

Dr. C. Eugene Riggs (St. Paul) gave the paper of the evening, entitled "The Dynamics of Personality."

DISCUSSION

Dr. Christison: I believe it would be hardly fair to let that masterly production go without comment. As an address on sociology, I have never heard anything to compare with it. It affects me particularly and peculiarly. I have been connected with social service work in St. Paul for a good many years. One of the subdivisions of our United Charities is a children's bureau, wherein we deal with delinquent as well as with normal children. The things that Dr. Riggs has told us are going to help me a very great deal in that work. I think he understands the psychology of childhood better than any one I have ever known.

Dr. Riggs, I want to thank you personally for that paper.

Dr. A. Schwyzer: I arose from the same impulse that Dr. Christison did; that is, to express our thanks for this splendid address. That is not an ordinary paper. That is an address to read over in our families and there is in it a good deal of matter that is good not only for doctors but for the members of their families and for laymen.

One great hope remains in what the Doctor told us, that most of these defects are not really hereditary but are really accidental happenings under peculiarly unfortunate circumstances and are individual conditions that are not propagated.

There is so much wholesome philosophy in this study that I think I speak in harmony with everybody present if I thank the Doctor heartily for what he gave us tonight.

Dr. H. L. Ulrich: Dr. Riggs raised the question of nature and nurture, which has been a subject for discussion ever since there have been biologists. For two years I have observed children going through the Child Guidance Clinic in Minneapolis. My work was to study the endocrinopathics in which we attempted to show that endocrine disturbance may have some bearing on behavior. We have not been able to demonstrate a single case in which the endocrines are factors in the misconduct of the children. In practically all cases one could say it was nurture rather than nature.

The meeting adjourned.

John E. Hynes, M.D.,
Secretary.

CASE REPORTS

Members are requested to report interesting and unusual cases for publication in this department. Many cases reported at hospital staff meetings and similar meetings are very instructive and worthy of publication.

STAPHYLOCOCCUS ENDOCARDITIS*

REPORT OF CASE

Victor K. Funk, M.D.
St. Paul

Mr. A. G., a German American, aged 56, was admitted to the hospital on May 25, 1925, complaining of: (1) shortness of breath; (2) pain over the precordium; (3) pain in the epigastrium; (4) dizziness; (5) neuritis.

Present Illness.—In September, 1924, the patient developed what he calls neuritis, which was manifested as a dull, nagging pain in his back, shoulders and neck. He was told at this time by his physician that his heart was normal. The pains moved from one part of the body to another, and obliged him to stop work. He saw several physicians, but obtained no relief from his pain. He then took a course of mud-baths, and at that time was told by a physician that he had leakage of the heart, but it was giving him no symptoms. In February, 1925, the patient had a severe attack of pain in the epigastrium which lasted three days. The pain was knife-like in character, and doubled him up. Two months prior to admission (March, 1925), while walking up a hill, he became very dizzy and had black spots before his eyes. He went to bed, but felt all right the next day, and remained well for one month. Then, one month prior to admission, he did some heavy lifting and following this became very short of breath. Since that time he has always been short of breath and has had a dull pain over the precordium. He frequently has had to sit up in a chair at night to get his breath and there has been some swelling of his feet and ankles.

Habits.—He has used tobacco to excess, but liquor moderately.

Past history.—He has had the usual diseases of childhood, but not scarlet fever, diphtheria, or rheumatic fever. Had never had any serious illness.

Physical examination.—The patient is well developed and well nourished, but very dyspneic, and has to sit propped up in bed. His lips and ears are very cyanotic. His tongue is coated and dry, and his breath has a bad foul odor.

Heart: Apex beat is visible as a wave in the fifth left intercostal space just outside of the nipple line. A systolic thrill is palpable over the whole precordium. On percussion the borders are 2 cm. to the right and 13 cm. to the left in the fourth intercostal spaces. On auscultation the heart sounds are entirely obliterated by a loud, blowing, systolic murmur, which corresponds in time with the radial

*From the service of Dr. J. S. Gilfillan, The Miller Hospital, St. Paul.

pulse. This murmur can be heard over the whole chest, but is loudest at the apex.

Abdomen: There is a slight tenderness in the upper right quadrant. Liver margin is palpable three fingers below the costal margin.

Extremities: No edema present.

Reflexes and sensation normal.

Blood pressure 100/70.

Laboratory findings.—Urine negative on two examinations.

Blood (June 22, 1925): Hgb., 65 per cent; r.b.c., 3,880,000; w.b.c., 12,900; p.m.n., 80 per cent; lymph, 20 per cent; r.b.c., normal.

Blood culture taken on June 22, 1925, showed 4 days later a luxuriant growth of staphylococcus aureus. The second blood culture, taken on June 26, 1925, again showed a staphylococcus aureus.

Clinical course.—The patient's daily temperature varied from 97 to 103.2 degrees. On a few days only his temperature did not rise above 99. The pulse rate ranged from 50 to 130, usually about 90, and was occasionally intermittent. He was at times irrational. On June 12, 1925, a red, painful nodule appeared on the tip of the left little finger. His breathing at times was very labored, and on July 9, 1925, he developed Cheyne-Stokes respirations. On July 17, 1925, he suddenly fell back in bed, and began frothing at the mouth as his respiratory passages filled with mucus. He remained in a comatose state for eight days and died on July 24, 1925.

Autopsy Report.—(Dr. Margaret Warwick.)

External appearance: One bright red area 2 cm. in diameter on left internal malleolus, and a similar smaller one 1 cm. in diameter on base of left toe.

Peritoneal cavity: No fluid, no adhesions; appendix retrocecal, bound down by adhesions.

Pleural cavities: No fluid. Firm fibrous adhesions over posterior surface on left.

Pericardial cavity: 25 c.c. of cloudy fluid in which were floating several large flakes of fibrin. Masses of fibrin are also found tightly adherent to wall of left ventricle.

Heart: Weight 430 g. The epicardium appears to be slightly opaque. Tightly adherent to it, and over the left ventricle are large flakes of fibrin which can be peeled off, leaving a smooth surface. The wall of the left ventricle is thickened to a moderate degree. The cavity of the left ventricle is moderately dilated. The aortic, tricuspid and pulmonary valves are all normal in appearance, but the mitral valve shows vegetations on two leaflets. One of these vegetations is a small compact mass on the leaflet just above the edge of the valve. Another is a very loose mass attached to the extreme edge of the leaflet and having with it a mass of clotted blood. Yet another small mass is attached at about the middle of the left auricular wall. All of these vegetations consist of very small masses of white, firm tissue, but with no evidence of ulceration. The heart muscle is rather soft in consistency and yellowish in color. At about the middle of the left ventricle is a white area about 1 cm. in diameter and evidently representing a fibrosed area. The coronaries show a very few slightly raised yellowish plaques which do not cause any appreciable narrowing or obstruction. The root of the aorta shows a very few slightly raised yellow patches.

Lungs: Left—dark red, crepitates throughout, showing on section a patchy red surface from which on pressure numerous droplets of pus exude. Right—solid and gray in color, showing on section grayish red consolidation with much pus escaping on pressure.

Spleen: Normal.

Liver: Cut surface shows a yellowish color with prominent tiny red mottlings, which stand out in sharp contrast.

Gall bladder, gastrointestinal tract and pancreas normal.

Adrenals: Soft centers.

Kidneys: Left, 160 g.; right, 140 g. Cortices thinned.

Bladder: Moderate amount of pus in contained urine. Mucosa red. No hypertrophy of prostate.

Head and neck not examined.

Cultures of heart's blood show a staphylococcus identical with that found before death.

Pathological diagnosis: (1) Staphylococcus bacteriemia; (2) acute vegetative endocarditis (mitral); (3) lobar pneumonia (right); (4) broncho-pneumonia (left); (5) acue fibrinous pericarditis; (6) acute cystitis; (7) healed pleuritis; (8) healed perisplenitis; (9) healed perihepatitis; (10) mild generalized arteriosclerosis; (11) fibrosis of heart veins.

Microscopic sections: Kidneys, adrenals, heart muscle, pancreas, all normal.

Lungs: Show the alveoli to be filled with pus cells in some areas; in other areas the alveoli are filled with desquamated endothelial cells, and some are filled with brown pigment (lobar pneumonia).

Bladder: Shows a much thickened wall in which is much new formed connective tissue with a very rich infiltration with lymphocytes and pus cells.

Liver: Shows the cords around the central veins to be thin and atrophic and to contain a large amount of brown pigment.

Prostate: Is atrophic and shows a very large amount of connective tissue.

Bronchial Node (left): Shows a loss of germinal centers and a rich infiltration with pus cells. There is also a large amount of brown pigment throughout.

A THALAMIC TUMOR DIAGNOSED HYSTERIA

George N. Ruhberg, M.D.
St. Paul

G. C., a man, 43 years of age, while feeding boards to a circular saw on June 3, 1924, was slightly injured. These boards were sixteen inches long and of equal thickness. The board that broke was not planed on one side, showing it was thinner than usual. The fingers of both hands were slightly scratched. He stated that he was a little disturbed over this accident, although it had happened before on numerous occasions. He resumed work and had worked a half hour or so, when he noticed another thin board. He did not feed it to the machine, but as he touched it, a queer sensation came over him. He felt dizzy and faint, and as if he had no left side to his body.

He slumped to the floor, and after a few moments tried to move his leg, but did not know where it was. His hand, while capable of slow motion, had no feeling. He soon became completely paralyzed over the entire left side of the body.

Examination in hospital by Dr. E. O. Giere showed his reflexes to be practically undisturbed, but he was unable to move the left side of his body, and he could not feel touch or pain over the entire area affected. On the second or third day he had regained partial use of the leg and arm, but the facial condition was replaced by pain and spastic movement, in which the left side could not be kept still, the arm moving in an irregular manner, so that taking an *x*-ray was impossible.

We were called in consultation, June, 1924, by Dr. Giere. The patient was abed and apparently deaf in the left ear. After a few moments of loud conversation, the voice was intentionally lowered and he understood perfectly. He complained that the sight of the left eye was not as good as that of the right, and tests revealed a narrowing of the field of vision in that eye. The facial muscles were normal in all movements, but he did not seem able to pucker his mouth to whistle. The tongue protruded straight. The grasp of the right hand was normal, but that of the left was diminished about one-half. There was no tremor. On walking, he dragged the left leg, but was able to support his weight on it. The reflexes were practically normal except the right plantar reflex, which was exaggerated and the left one absent. His Wassermann was reported positive at the hospital. He could not feel touch or pin-prick over the entire left side, including the face. The diagnosis made at this time was hysteria.

We saw him again on August 18th, when he came under our care. He walked with a limp of the left leg, and he had about a fifty per cent diminution of the strength of the left hand. He could, with difficulty, cross the left leg over the right. When he stood with his feet together he swayed to the left. The knee-jerks were somewhat increased, but no Babinski was present. The sensation to pain was absent over the entire left side. With regard to his hand, he could slowly perform all fine finger movements, but in a clumsy manner, and not with the same facility as with the right hand. His eye grounds were normal, and no diminution in the field of vision was present. Under treatment his anesthesia seemed to vary, in that points where he was insensitive, he could feel perfectly. The sensory tests varied so much at different times that it was difficult to definitely form an opinion as to their reality.

He passed from our care at this time to that of another neurologist in St. Paul, who concurred with our diagnosis. After this he was seen by other neurologists, both on his behalf and also for the insurance company, and also was seen by numerous other doctors, who were not neurologists. The diagnosis of traumatic hysteria was made by practically every doctor who saw him.

His condition became gradually worse, until June, when he was admitted to my service at the Ancker hospital. At this time his pupils did not react to light or accommodation. His left pupil was larger than the right. He had weakness of the left extremities. Reflexes on the left were hyperactive. A positive Babinski on the left and ankle clonus were present. He had incontinence of the bladder, and his mental state was stuporous, so that it was impossible to carry out a good test of superficial sensation. His blood Wassermann and spinal fluid tests were negative. The mistake in diagnosis, of course, was here apparent, as many signs of a central organic involvement were present. He died suddenly after admittance, and a post-mortem was performed by Dr. Noble. A hemorrhagic glioma, involving the right thalamic area and causing pressure on the internal capsule, was present. It was not encapsulated, was red and hemorrhagic in appearance, and soft and friable in consistency.

Two factors are present which can account for the common error in diagnosis. The first was that the case was a chronic one, extending over months in duration, during which time he never was under the observation of one man for over a short time. The other was the history of sudden onset, coincidental with the accident. The history given by us is as the man told it at the time of the first examination. Perusal of the court records shows that he gave a slightly different history on the stand, in that he stated he was neither surprised nor frightened at the time of the accident. By tracing the man from one doctor to another, and combining their observations, we find that this man at one time or another presented the following: hemichorea, hemiataxia, hemitremor, hemiathetosis, hemianesthesia, hemianopsia, and an exaggerated reaction to pain, involving chiefly the left shoulder. It was a typical thalamic syndrome. One neurologist was of the opinion that the man suffered from hysteria at first, which later merged into that of the organic syndrome. Our opinion was that he had the tumor before the accident, and that, due to its hemorrhagic nature, a sudden hemorrhage, sufficient to cause symptoms in this area, occurred coincidental with the accident.

The symptoms coming on abruptly at the same time as the accident, has made this case not only interesting as a diagnostic problem but also from a medico-legal aspect. It presents an example of a brain tumor which was repeatedly diagnosed traumatic hysteria by several qualified neurologists, as well as numerous general practitioners, and for that reason is reported.

But most important to us, is the security against smallpox existing in the state in which we live. This security depends upon the degree to which vaccination is practiced, and this in turn depends upon the enlightenment of its people and the existence and enforcement of vaccination laws. Some states which report such a large number of smallpox cases and deaths are among those which are most afflicted by the organized activities of the anti-vaccinationists. For example, vaccination is unpopular in Minnesota. The propaganda of the anti-vaccinationists has been successful since 1903 in keeping any laws requiring rigid statewide vaccination off the statute books, nor can vaccination be enforced in times of epidemics. The result is what one might expect. With a record of rarely less than 1,200 reported cases, and over 9,000 reported cases in 1921, Minnesota furnishes a striking example of what a state's smallpox history may be when vaccination is refused by its people.—*Am. Assn. for Medical Progress.*

PROGRESS

Abstracts to be submitted to Section Supervisors.

Members are urged to abstract valuable articles which they run across in their reading and send the abstracts to the physicians in charge of the respective sections. In order to avoid duplication it would be well to communicate with one of the section supervisors before the article is abstracted.

MEDICINE

SUPERVISORS:
F. J. HIRSCHBOECK,
FIDELITY BLDG., DULUTH
THOMAS A. PEPPARD,
LA SALLE BLDG., MINNEAPOLIS

DISEASE OF THE CORONARY ARTERIES: M. H. Nathanson (Abstract from the Am. Journ. of the Med. Sciences, August, 1925). Nathanson reports that out of 849 autopsies in 1923, from the Department of Pathology, the University of Minnesota, there were 28 deaths attributed to coronary sclerosis. Of these, 27 were above the age of forty. In this same group there were 70 cases of malignancy of various types, indicating the relative frequency of coronary sclerosis. On the other hand, 12 of the coronary cases were from the coroner's service, which would tend to exaggerate the frequency to a certain degree.

The author gives credit to Herrick for the newer conception of coronary disease and the various types of its clinical course. The author has endeavored to describe a clinical picture based on the material for study from the Department of Pathology, University of Minnesota, and comprises records of 113 cases. All these included severe coronary disease with narrowing and obliteration of one or more large vessels and fibrosis or softening of the heart muscle. No case was selected in which coronary disease was not considered the chief cause of death.

The proportion of males to females was the ratio of 6 to 1, and 65 per cent of the cases occurred between the ages of fifty and seventy years.

The most common symptom was pain, varying from an uncomfortable, heavy feeling, to an intense viselike sensation. The pain was of the type usually associated with coronary disease and angina pectoris, such as precordial or epigastric distress, with radiation into the upper extremities.

Attacks of respiratory distress were also frequently observed.

In 60 per cent of the cases the heart size was increased. Congestive failure was present in about 40 per cent, as evidenced by pulmonary and hepatic congestion.

Four types of clinical features were noticed:

(1) The heart normal in size, with normal blood pressure, and absence of cardiac insufficiency and congestive failure.

(2) Heart of normal size, with symptoms of congestive failure as well as cardiac insufficiency, but normal blood pressure.

(3) Cases of enlarged heart, with increased blood pressure, but with absence of congestive failure or heart insufficiency.

(4) Cases with cardiac enlargement, increased blood pressure, congestive failure, and cardiac insufficiency.

Of the 113 cases, 24, or 21 per cent, showed coronary thrombosis.

Thrombosis may be considered as a complication of coronary sclerosis, rather than as a separate clinical entity.

DR. F. J. HIRSCHBOECK.

CLINICAL VALUE OF SOME RECENT TESTS FOR LIVER FUNCTION: Howard F. Shattuck, M.D., John C. Browne, M.D., and Majorie Preston, A.B., New York (Abstract from the Am. Journ. of the Med. Sciences, October, 1925). The authors made tests in the various clinical conditions with the more commonly used liver function tests, including the Rowntree-Rosenthal test, icterus index, the van den Bergh and Fouchet tests.

In their experience the Fouchet test proved unreliable for detecting minor grades of bilirubinemia. The van den Bergh test had every advantage, and none of the disadvantages of the Fouchet test. The van den Bergh test was of particular value in the specific qualitative test for bile pigment to control readings of the icterus index in patients with latent jaundice. It also aids in distinguishing between hemolytic and obstructive jaundice.

The icterus index test is the most useful single functional liver test, for the following reasons:

(a) Its ease and rapidity of performance.

(b) In the diagnosis of cases of cholecystitis and cholelithiasis without clinical jaundice, in which event a latent bilirubinemia is oftentimes discovered.

(c) It helps to distinguish between obstructive jaundice due to malignancy and catarrhal jaundice by showing whether the jaundice is increasing, diminishing or stationary.

(d) In the diagnosis of hepatic disease per se it runs rather parallel with the dye retention test.

(e) It may serve as a guide to the treatment of syphilis with arsenicals, and the toleration of the liver to the drug.

(f) It will determine whether obstructive jaundice has been relieved by operation.

(g) It helps to differentiate the primary from the secondary anemias.

(h) It may indicate the degree of cardiac decompensation.

The Rowntree-Rosenthal test is thought to be of supplementary value, and is of greater value than the icteric index in estimating liver efficiency in cirrhosis and malignant metastases. It also is of value in estimating the operative risk in case of surgical intervention. Its greatest value lies in the diagnosis of the liver insufficiency in patients without jaundice.

DR. F. J. HIRSCHBOECK.

PEDIATRICS

SUPERVISORS:
CHESTER A. STEWART,
LA SALLE BLDG., MINNEAPOLIS
ROY N. ANDREWS,
MANKATO CLINIC, MANKATO

PROGRESS IN PEDIATRICS — REVIEW OF THE LITERATURE ON INFANT FEEDING FOR 1924: Rudolph Duryea Moffett, M.D. (Amer. Jour. of Diseases of Children, Nov., 1925). Richardson has proved by statistics that the breast-fed baby has five times the chance for life and health as the artificially fed infant. The best means of stimulating secretion is by nursing or by rhythmic expression of the milk by the thumb and forefinger, applied just back of the nipple. The so-called colic in breast-fed infants is often a hunger symptom, which may be verified by putting the child back on the breast.

One of the important steps in solving the problem of breast feeding is regular weighing of the child before, during and after the nursing; this indicates whether the child is getting enough milk. Breast milk may be increased by giving both breasts at each nursing, by increasing the intake of fluids, by proper exercise and by adequate rest.

Koeppe agrees with many authors that the qualitative analysis of milk is valueless. Constipation, diarrhea, loss of weight, pallor, feeble pulse, subnormal temperature and alterations in the blood picture are known indications of under-feeding.

Artificial Foods.—Sweetened condensed milk and powdered milk, for the feeding of infants in the tropics, had been investigated by Deeks. These canned foods proved satisfactory for normal growth in 90 per cent of the babies. The old theory that protein is difficult to digest now seems unreasonable.

Struthers, Gleich, Field, Mulherin and Marfan and his co-workers have considered the use of acidified milk, particularly for young infants with nutritional disturbances. This preparation affords an excellent means for treating athreptic infants and may be given in concentrated feedings, which overcomes much of the vomiting. After the administration of acidified milk, the acid of the duodenum increases in a half hour and persists for one hour. Buttermilk produces no increase in the acidity of the infant's duodenal content.

Butter Flour Feedings. — In reporting their experience with the Czerny-Kleinschmidt butter flour mixture, Schumacher, Wisch and Greenwald are convinced that butter flour is of value in feeding feeble and premature infants as well as healthy ones. For children with acute nutritional disturbances manifested by diarrhea, fever and loss of weight, the high fat feeding seems to intensify the condition.

Peristaltic Function. — Rogatz found that the infant's stomach reacts differently to fluids and to semisolids. When fluids are given, the stomach assumes either a pear or oval shape, horizontal in position, extending beyond the middle with a large air bubble. With food that is very thick, the infant's gastric organ contracts down to a small circular shape, barely reaching the middle line and never extending beyond it, with little or no air bubble present. These facts prove the necessity of more concentrated food in place of fluid feeding.

Metabolism.—In a study of the metabolism in undernourished infants, Fleming and Hutchison consider as causes of failure to gain: (1) defective absorption of food, (2) defective utilization of absorbed food in the tissue, and (3) excessive combustion of absorbed food.

R. N. ANDREWS, M.D.

PROGRESS IN PEDIATRICS — IMPORTANCE OF CONSIDERING THE BODY MECHANICS OF CHILDREN: Lucy Porter Sutton, M.D. (Amer. Jour. of Diseases of Children, October, 1925). In examining a child the physician investigates carefully the throat, the heart, the lungs and the abdominal organs. Too frequently he completely ignores the skeletal system unless there is a marked lordosis, an obviously protuberant abdomen or flat feet. Good body mechanics means that the body is maintained in such a position that there is no undue strain on any set of muscles or organs.

At the basis of poor posutre lies the fact that our bodies were originally designed for the four-legged position, and we have not yet fully evolved a mechanically perfect two-legged state. Once the abdomen has started to sag, the other factors in poor body mechanics develop, namely, the flat chest, forward head, tilted pelvis, round shoulders sprung knees and knock knees. When fatigue is added to these conditions, a vicious cycle is established—the more tired a person becomes the worse he stands; the worse he stands the more tired he becomes because of the undue and unqual muscle strain.

In children there are certain conditions which are definitely relieved or cured by correction of the body mechanics. These are: (1) Constipation, unless there is an organic lesion. (2) We frequently see patients who complain of vague abdominal discomfort. Many are completely relieved by correction of the body mechanics. (3) Cyclic vomiting. Postural treatment and avoidance of overfatigue are logical aids in preventing the attacks. (4) Enuresis. This is almost always a difficult condition to treat, but some children respond surprisingly to posture work.

Many of the children found in nutrition classes fall into the slender visceroptotic class and are frequently the ones who prove most refractory to treatment. When such children have their body mechanics improved there is almost always a gradual but steady gain in weight. Scoliosis and pronated feet are sometimes secondary to poor posture.

Besides these definite conditions, which are frequently corrected by improving the body mechanics, it is surprising to see the improvement in the general condition of the children.

Treatment.—The first essential in attempting to correct the body mechanics of a child is to explain to the parents and nurse the reasons for the need and to demonstrate the position of the body, which is most desirable. The mat-

tress of the bed should be firm, and no pillow should be used. If an underwaist is worn, the straps should go over the base of the neck rather than over the tips of the shoulders, as this is often an element in bringing the shoulders forward. Bloomers with elastic around the waist are bad. Side garters fastened to an improperly fitted waist exert a strong influence in bringing the shoulders forward. Children should have chairs which are of the proper size. In the case of small children and those with bad posture and malnutrition, it is advisable to insist on a rest in an overcorrected position, preferably after each meal.

The simplest position to take is lying on the back on a firm flat surface such as the floor, with a small roll or pillow under the lower part of the thorax. The hands are placed behind the neck, and the shoulders and elbows rest on the floor. The exercises used should have two aims: (1) to teach the child conscious control of the muscles, and (2) to strengthen the muscles so that when the proper position has been learned the child will be able to maintain it.

The results obtained are well worth the time and patience required.

R. N. Andrews, M.D.

A FREQUENT CAUSE OF DYSPEPSIA IN BREAST-FED INFANTS: Kirsten Utheim Toverud, M.D. (Amer. Jour. of Diseases of Children, November, 1925). Quite frequently, one observes breast-fed infants who, after doing well for the first few weeks or months of life, stop gaining in weight, and become restless and irritable. Vomiting, occasionally severe, may be the predominating symptom. Ultimately these infants lose weight, and unless proper treatment is instituted they become distinctly athreptic.

In the author's experience, overfeeding with breast milk is a rare cause of dyspepsia. Faulty composition of mother's milk does not seem to be a factor in the causation of the symptoms of dyspepsia. In a series of cases, the breast milk was analyzed and the figures for sugar, fat and protein were not found to differ from those of the milk of mothers whose babies were thriving.

In a group of thirty-three infants, in whom the symptoms enumerated were present, it was found in each instance that the mother was secreting an insufficient amount of milk. When suitable supplementary feelings were given to these infants, a prompt disappearance of the symptoms occurred regularly. Partial inanition may lead to a variety of symptoms, including weight loss, weakness and constipation, as well as dyspeptic symptoms.

Inanition is far more serious in the case of an infant than in an older person. The infant has a higher energy requirement as well as a greater need for material for building up new tissues. Furthermore, the infant does not store a sufficient amount of reserve food to supply its needs for any considerable time.

Nausea and vomiting are frequent symptoms of hunger, even in adults. Infants, in general, vomit more readily than older people, and with the marked hunger phenomena that they exhibit it is not surprising that vomiting should occur.

R. N. Andrews, M.D.

ROENTGENOLOGY

SUPERVISORS:
LEO G. RIGLER,
MPLS. GEN'L HOSPITAL, MINNEAPOLIS
A. U. DESJARDINS,
MAYO CLINIC, ROCHESTER

REPORT OF 5,000 GASTRO-INTESTINAL EXAMINATIONS BY THE ROENTGEN RAY: Merrill (Am. Jour. Roent., V. 14, p. 310, Oct., 1925). 818 of these cases were explored surgically and these findings, the clinical diagnosis, and the *x*-ray diagnosis are tabulated.

There was a total error of 4 per cent in this group of cases as determined by operation. A positive error, that is the diagnosis of lesions not found at operation, of 1.2 per cent is reported. No gastric cancer or ulcer, found at operation, was missed by the roentgen examination. Three cancers of the colon were missed, due no doubt to failure to employ the barium enema in these cases.

In 551 cases, showing ptosis only roentgenologically, a clinical diagnosis of peptic ulcer had been made in 28 per cent and of cancer in 8 per cent. Only 15 of these were explored, but in none was gastro-intestinal pathology found, confirming the *x*-ray diagnosis in every case.

In duodenal ulcer, the roentgen findings were correct in 92 per cent of the cases, only one-half of these having been recognized clinically. In gastric ulcer, the roentgen diagnosis was confirmed in 93 per cent, the clinical diagnosis only in 56 per cent. In gastric cancer, the comparison is even more astonishing. A correct roentgen diagnosis was made in 95 out of 97 cases, 98 per cent; clinically 38 per cent were diagnosed cancer of the stomach, 18 per cent cancer of undetermined location, and in 44 per cent the diagnosis was either incorrect or not made.

Other outstanding figures were the correct diagnosis of appendicitis in 34 of 35 cases and of gall bladder disease in 32 of 34 cases. In 127 examinations of the esophagus, the roentgen diagnosis was confirmed in every case, either by esophagoscopy or operation. Clinically, 18 of 70 cases of obstruction, 13 cases of cardiospasm, and 6 cases of diverticulum went unrecognized. On the other hand, in 24 of 35 cases roentgenologically negative, a clinical diagnosis of obstruction had been made.

The report is carefully prepared and comes from the Massachusetts General Hospital. It emphasizes in striking fashion the vital importance and almost unbelievable accuracy of the roentgen diagnosis in diseases of the gastro-intestinal tract.

Leo G. Rigler, M.D.

IN BEHALF OF THE RELIABILITY OF THE NICHE IN GASTRIC ULCER DIAGNOSIS: M. Haudek (Fort. a. d. Geb. d. Roentgen., V. 33, p. 5, Sept., 1925). In gastric ulcer diagnosis, the niche is a perfectly reliable sign, if the entire characteristic symptom-complex is present. However, it must be emphasized that such a diagnosis, with the

niche as its basis, is safely made only in connection with lesions of the pars media of the stomach.

Criticisms regarding the relative value of the niche as a diagnostic sign are entirely based on faulty interpretation, as may be shown by a careful analysis of the cases reported. Thus there are frequent failures in the attempt to demonstrate the ulcer operatively, even though the roentgen picture is typical, this being due to incomplete exploration of the comparatively inaccessible posterior stomach wall. Lack of differentiation of the second portion of the duodenum from the gastric contour; calcified tumors or lymph nodes; left-sided nephrolithiasis; diverticulum of the duodeno-jejunal angle; true congenital gastric diverticuli—all these constitute pitfalls for faulty interpretation.

When the lesion is located on the lesser curvature of the stomach, the niche of gastric ulcer can always be differentiated from that of carcinoma, if the observations are very carefully made. The observer must here keep in mind the differential findings of ulcer and carcinoma in this particular region, with special reference, (1) to the contour and degree of irregularity, and the nature of the peristaltic waves of the lesser curvature; (2) to the base, size, shape, permanency of and retention in the projecting shadow; (3) to the width, the character of the outline, and the general direction of the incisura on the greater curvature.

With the statistics of Orator, as found in Eiselberg's clinic, as a basis, malignant degeneration occurs in 2 per cent of ulcers of the body of the stomach, in 30 per cent of pre-pyloric and in 0 per cent of duodenal ulcers. Diagnosis of such degeneration is dependent on the recognition of the slightest variation from the definite ulcer characteristics, coincident with the demonstration of signs simulating carcinoma.

WALTER H. UDE, M.D.

EYE, EAR, NOSE AND THROAT

SUPERVISORS:

VIRGIL SCHWARTZ,
PHYS. & SURG. BLDG., MINNEAPOLIS

E. L. ARMSTRONG,
FIDELITY BLDG., DULUTH

TREATMENT OF PHLYCTENULAR INFLAMMATIONS OF THE EYE: Dr. Shinichi Funaishi (Mukden, Manchuria, American Journal of Ophthalmology, August, 1925, p. 618). Whereas various investigators had injected tuberculin or tubercle bacilli into animals and had later instilled some of the same substance into the conjunctival sacs of these animals and so produced phlyctenular inflammation of the eye, it had been concluded that the condition was essentially a tuberculous one. However, the writer has produced identical results by using instead of tuberculin, a staphylococcus vaccine, or a solution of tyramin hydrochloride or of casein. The substance common to all these and to tuberculin or the tubercle bacillus, is a protein foreign to the animal, which causes the general sensitivity of the animal (to these proteins) to react with a local manifestation in the eye. Therefore when a patient is sensitized to foreign proteins, as is the case in the scrofulous or exudative diathesis, or in gastrointestinal intoxication, in absorption of destroyed products of secretion in eczema, and in bacterial infections which deposit proteins in the blood, if bacterial or other foreign proteins should invade the conjunctival sac, a phlyctenular lesion may readily result.

Cow's milk may be used to improve the body resistance to the reactions of various proteins. Injection of one to four c.c., according to age, of fresh cow's milk, boiled three or four minutes, into the gluteal muscles produces only a temporary rise in temperature, and may be repeated every three or four days. The secondary fever diminishes each time until it does not exceed 99° F.

VIRGIL J. SCHWARTZ.

STATISTICAL STUDIES OF THE CHILDREN IN THE CHICAGO PUBLIC SCHOOLS FOR THE DEAF: George E. Shambough, M.D., assisted by Alice K. Hall, M.D., W. E. Hagens, M.D., and J. W. Halderman, M.D. (Abstract from the Archives of Oto-Laryngology, November, 1925, p. 417). Two hundred ninety children from three public schools for the deaf in Chicago were examined.

An effort was made to determine the relationship between nose and throat conditions and the existing ear trouble. Only in a few cases could correctable nose and throat conditions be held accountable for the development of the ear trouble. The type of ear trouble was that of the internal ear or nerve deafness. Many had had tonsils removed previously without benefit to ear conditions.

This type of ear trouble could probably not have been influenced much by any efforts at prevention.

It is a fallacy that adult deafness is due to adenoids and hypertrophy of tonsils during childhood.

Congenital deafness numbered 119 cases. There was history of deafness in family in only 40 of the cases. Acquired deafness cases numbered 145. Of these 56 at the onset had otitis media. The deafness of this group was practically always referable to pathology of the labyrinth.

Meningitis was a cause in 25 cases. Scarlet fever was the cause in 14 cases. Mixed infection was the cause of 11 cases. Pneumonia was the cause in 9 cases. Diphtheria was the cause in 9 cases. Measles was the cause in 8 cases. Otitis media was the cause in 6 cases. Typhoid fever was the cause in 4 cases. Influenza was the cause in 3 cases. Fracture of the base of the skull was the cause of 3 cases. Whooping cough was the cause of 2 cases, and infantile paralysis was the cause of 3 cases. Mumps was the cause in 1 case.

HEREDITY

Under this heading forty-nine cases are reported. Some of these were due to syphilis, but in most of them the cause was not definitely determined.

E. L. ARMSTRONG, M.D.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

LICENTIATES OF OCTOBER, 1925

BY EXAMINATION

NAME	MEDICAL COLLEGE	ADDRESS
Armstrong, Harry G.	U. of Louisville, M.D., 1925	St. Paul, St. Luke's Hosppital
Balch, Franklin Greene, Jr.	Harvard, M.D., 1923	Rochester, Mayo Clinic
Bowles, John Herschel	Rush, M.D., 1925	Rochester, Mayo Clinic
Decker, Walter Joseph	Wash. U., Mo., M.D., 1923	Rochester, Mayo Clinic
Fenger, Ejvind Palmer K.	U. of Minn., M.B., 1924	Oak Terrace, Minn.
Fink, Leo William	U. of Minn., M.B., 1924	Minneapolis University Hospital
Giere, Richard Waldorf	U. of Minn., M.B., 1925	St. Paul, Hamm Building
Hedemark, Truman Albert	St. Louis U., M.D., 1925	St. Paul, Ancker Hospital
Heiberg, Emmett Anderson	U. of Minn., M.B., 1925	St. Paul, Ancker Hospital
Lindsey, Maude Louise	Wash. U., Mo., M.D., 1924	Rochester, St. Mary's Hospital
Olson, Chester Jerome	U. of Minn., M.D., 1925	Belle Plaine, Minn.
O'Reilly, Bernard Eugene	St. Louis U., M.D., 1925	St. Paul, Ancker Hospital
Paul, Louise Mary	U. of Minn., M.B., 1925	Minneapolis, 504 Ridgewood Ave.
Pierce, Alano E.	U. of Minn., M.B., 1924	Minneapolis, General Hospital
Shepard, Chas. Edward	U. of Minn., M.D., 1924	Le Mars, Iowa
Sundberg, Rudolph Herbert	U. of Neb., M.D., 1925	St. Paul, Ancker Hospital
Wahlquist, Harold F.	U. of Minn., M.B., 1925	Minneapolis, 3310 Fremont Ave. S.
Wall, Mark Henry	U. of Minn., M.B., 1924	Minneapolis, 510 Essex St.

BY RECIPROCITY

Bannick, Edwin George	U. of Ia., M.D., 1920	Rochester, 426 6th St. S. W.
Beach, Watson	Detroit Col. Med., M.D., 1924	Rochester, Mayo Clinic
Burling, Fred Temple	Rush, M.D., 1923	Decorah, Ia.
Chumley, Charles Lawrence	U. of Tenn., M.D., 1924	Rochester, Minn.
Cumming, John Frederick	U. of Toronto, M.B., 1922	Abercrombie, N. D.
Dugan, Lawrence F.	Marquette, M.D., 1924	Milwaukee, Wis., 320 Sycamore
Dworsky, Samuel David	U. of Minn., M.D., 1924	Minneapolis, 1228 Upton Ave. N.
Fahr, Geo. Edmeston	Wuerzburg, Ger., Dr. of Med., 1909	Minneapolis, 400 5th St. S. E.
Fawcett, Wm. Crozier	Western Ontario, M.D., 1901	Starkweather, N. D.
Greenlee, Daniel Paul	U. of Pittsburgh, M.D., 1924	Rochester, Minn.
Horton, Bayard Taylor	U. of Va., M.D., 1922	Rochester, Minn.
Hutchinson, Dorothy Wilder	U. of Pittsburgh, M.D., 1924	St. Paul, 2007 Portland
Hyslop, Orton Chas.	Northwestern, M.D., 1916	Marble, Minn.
Jacobs, Minard Friedberg	U. of Mich., M.D., 1923	Rochester, Minn.
Jensen, Julius	London, L.R.C.P., M.R.C.S., 1923	Starbuck, Minn.
Judge, Walter Thomas	U. of Iowa, M.D., 1924	Graceville, Minn.
King, Harry Thomas	Marquette, M.D., 1924	Minneapolis, 2607 17th Ave. S.
Lapierre, Jean Thos.	Creighton, M.D., 1924	Minneapolis, 303 E. Hennepin
McNaugher, Wm. McMillan	U. of Pa., M.D., 1924	Rochester, Minn.
Meng, Eleanor Lovejoy	Hah. Chic., M.D., 1910	Fergus Falls, Minn.
Meng, William Lucius	Hah. Chic., M.D., 1910	Fergus Falls, Minn.
Ortman, John Wessel	Creighton, M.D., 1924	Pierz, Minn.
Pugliese, Francis Michael	U. of Pa., M.D., 1923	Rochester, Minn.
Stuhler, Louis George	U. of Ia., M.D., 1906	Rochester, Minn.
Viecelli, James D.	U. of Colorado, M.D., 1923	Rochester, Minn.

NATIONAL BOARD CERTIFICATE

Anderson, Ernest Raymond	Rush, M.D., 1925	Minneapolis, 808 E. Franklin Ave.
Baumgartner, Conrad John	U. of Nebr., M.D., 1923	Rochester, Minn.
Eskew, Don Carlos	U. of Va., M.D., 1924	Rochester, Minn.
Fishback, Frederick Coleman	Harvard, M.D., 1922	Rochester, Minn.
Hurt, Holcombe Harris	U. of Va., M.D., 1924	Rochester, Minn.
Schutz, Elmer Scenas	Rush, M.D., 1925	Mountain Lake, Minn.
Whitten, Merritt Bryant	U. of Oregon, M.D., 1924	Rochester, Minn.

BOOK REVIEWS

BOOKS RECEIVED FOR REVIEW

MINNEAPOLIS, ST. PAUL AND SAULT STE. MARIE RAILWAY SURGICAL ASSOCIATION TRANSACTIONS, 1923. 235 pages. Published in Minneapolis, 1925.

INTRAVENOUS THERAPY: ITS APPLICATION IN THE MODERN PRACTICE OF MEDICINE. Walton Forest Dutton, M.D. 2nd revised and enlarged edition. 594 pages. 64 halftones and engravings. Cloth, $6.00. Philadelphia: F. A. Davis Company, 1925.

A TEXTBOOK OF PHYSIOLOGY. Wm. D. Zoethout, Ph.D., Professor of Physiology, Chicago College of Dental Surgery (Loyola University) and Chicago Normal School of Physical Education. 2nd edition. 616 pages. Illustrated. Cloth, $4.50. St. Louis: C. V. Mosby Company, 1925.

THE THERAPY OF PUERPERAL FEVER. Privatdozent Dr. Robert Koehler, formerly assistant of Gynecological Department of Krankenhaus Wieden, Vienna. American edition prepared by Hugo Ehrenfast, M.D., F.A.C.S. 276 pages. Illustrated. Cloth, $4.00. St. Louis: C. V. Mosby Company, 1925.

THE SURGERY OF PULMONARY TUBERCULOSIS. John Alexander, B.S., M.A., M.D., Assistant Professor of Surgery in the Medical School, University of Michigan. Introductions by Hugh Cabot, M.D., C.M.G., H.D., F.A.C.S., and Edward R. Baldwin, M.A., M.D. 356 pages. Illus. with 53 engravings and 12 plates. Philadelphia and New York: Lea & Febiger, 1925.

EYE, EAR, NOSE AND THROAT MANUAL FOR NURSES. Roy H. Parkinson, M.D., Visiting Oculist and Aurist to St. Joseph's Hospital, San Francisco, California. 207 pages. Illus. Cloth, $2.25. St. Louis: C. V. Mosby Co., 1925.

The purpose of this book is to supply a system of teaching eye, ear, nose and throat for the classroom in nurses' training schools. The author's correct contention is that in teaching nurses you are not training specialists and he has made the diction free and readable, nomenclature is simplified and technical discussions have been avoided. Illustrations from pen sketches by the author are clear and understandable and furnish good copy for "chalk talks" in lectures. The second part deals with operating-room technic and illustrations of the layout of instruments for the various operations. Problems of the public health nurses are discussed in the third part. This book as a whole is to be commended as a basis for lectures to the lay mind—how much to say or how little, how to say it, how to illustrate and make clear what we are saying.

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